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Ecological Effects of Pesticides on Non-Target Species

Cornell Univ, Ithaca, N Y Dept of Entomology and Limnology

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Ecological Effects of PESTICIDES on Non-Target Species

EXECUTIVE OFFICE OF THE PRESIDENT
OFFICE OF SCIENCE AND TECHNOLOGY
JUNE 1971

David Pimentel, Department of Entomology and Limnology,
Cornell University, Ithaca, New York

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June 1, 1971

This volume was prepared at the request of the Office of Science and Technology in the Executive Office of the President, by Dr. David Pimentel, Department of Entomology and Limnology, New York State College of Agriculture, Cornell University.

We commissioned the study because it was evident that there was no single source of data on the environmental effects of pesticides. Furthermore, some data that existed and were quoted by various individuals to support a particular view had never been formally published, in some cases because the experimental methods were subject to question and results had not been confirmed by more discriminating later experiments. We intended this volume to be a comprehensive compilation of data, screened with some care to eliminate most unsubstantiated reports, so that discussions of environmental effects could concentrate on legitimate differences in value judgments rather than arguments about the validity of the scientific facts.

Ecological Effects of Pesticides on Non-target Species is a comprehensive compilation of published data. The judgments on what to include and what not to include are those of the author, though many individuals in the Federal government offered suggestions and critical review during its preparation. It is published by the Office of Science and Technology as received from the author in the belief that it will be a very useful contribution to the public discussion of pesticides. Publication by the Office of Science and Technology does not imply responsibility for completeness or accuracy of the information included.

Edward E. David Jr.

Edward E. David, Jr.
Director

Contents

		Page
ACKNOWLEDGEMENTS		iv
PART I	Introduction	1
PART II	Insecticides	[illegible]
PART III	Herbicides	85
PART IV	Fungicides	127
PART V	Pesticide Residues in the Environment	163
PART VI	An Evaluation and Summary	177
APPENDIX A	Abbreviations Used	183
APPENDIX B	Common and Scientific Names of Animals and Plants	185
APPENDIX C	Index of Pesticides: Common and Chemical Names	189
APPENDIX D	Subject Index	207

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PART I

Introduction

Clearly, it is vital to the well-being of man that he continue to sustain high production in agriculture while at the same time making certain to maintain a viable life system. Pesticide chemicals have played a significant part in increasing agricultural production and productivity. Unfortunately, this increased production has been paralleled by an increase in pollution resulting in part from agricultural practices, including pest control.

In 1970 nearly a billion pounds of some 900 registered pesticides were applied (more than 50 percent for farm use) throughout the United States for pest control. This large quantity of pesticide was aimed primarily at about 9,000 pest species of plants and animals. As expected, many of the other 200,000 species of plants and animals present in the environment were either directly or indirectly affected by these widespread pesticide applications.

Pest control is necessary for the adequate production of food and fiber. But also important to us are the many wild plants and animals that comprise and maintain the life system in which we exist. Evidence suggests that the great majority of the 200,000 non-target species are a necessity for our survival, for we cannot survive with only our crop plants and livestock.

This report summarizes the available evidence concerning the impact of pesticides (insecticides, herbicides, and fungicides) on individuals, populations, and communities of species and the magnitude of the reported damage to the diverse processes of living systems. For each named pesticide, such as 2,4-D, pertinent information has been presented about its influence on non-target mammals, birds, fishes, amphibians, mollusks, arthropods, annelids, plants, and microorganisms, as well as its biological concentration in food chains and persistence (when the information is available). No comment will be made concerning a reviewed paper, but an assessment of the total evidence relative to the dangers to the ecology of populations, communities, and ecosystems will be presented in Part VI.

This review is selective, aiming to include only scientifically valid investigations. In determining whether a report was to be included in the review several questions were asked: What was the dosage or application rate of the pesticide administered to the non-target species or their habitat? Were there adequate controls? Was sampling adequate? There were discrepancies in results between some reports reviewed, but these were included when sound experimental methods were employed in the investigation. The reason for the differences were not always evident from the papers themselves, but obviously there was either some variation in procedures or the presence of some chance event not evident to the investigators. These reports, however, are of considerable value because they do provide an idea of the range of response to a pesticide by non-target species. Studies reported in progress reports at research laboratories were cited only when the results could be verified and approved by investigators and/or their close associates.

The LD_{50}'s derived from single oral doses of chemicals should be regarded only as guides or benchmarks because many other factors may alter toxicity in the environment, and evidence suggests that compounds which are more poorly absorbed or cumulative in action are better tested by long-term feeding or by repeated doses. Consequently, these LD_{50}'s are included with the warning that direct comparisons between persistent and more readily degradable pesticides may be misleading.

In this review laboratory toxicity data cannot be directly related to possible field exposures. Although information was available on concentrations applied in the field, often no data were presented on the quantities picked up by the organism. Directly related to the amount of pesticide entering and affecting organisms like fish, are factors of temperature, hardness, and movement of water. In the investigations involving persistence, seldom was there a stated criterion for "disappearance," e.g., 50 or 90 percent loss of the pesticide.

Overall, I am less than pleased with the information available, and the review clearly points out the desperate need for intensive investigation concerning the ecological effects of pesticides on non-target species.

PART II

Insecticides

ABATE

Mammals

The LD_{50} for male rats was 151 mg/kg (Tucker and Crabtree, 1970) and for rats (no sex given), 9,000 mg/kg (FCH, 1970) to abate when the animals were fed the stated dosages orally. No explanation is available for the large discrepancy.

Birds

The LD_{50} for mallard ducks was 80 to 100 mg/kg; for young pheasants, 31.5 mg/kg; for young chukar partridges, 270 mg/kg; for young coturnix, 84.1 mg/kg; for pigeons (*Columba livia*), 50.1 mg/kg; and for house sparrows, 84.4 mg/kg to abate when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was 1,400 to 1,600 ppm; for pheasants, 150 to 170 ppm; for bobwhite, 90 to 110 ppm; and for coturnix, 280 to 370 ppm of abate in diets of 2-week-old birds when fed the treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

When chickens were fed abate at a dosage of 125 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The 48-hour LC_{50} for brook trout exposed to abate was 1,500 ppb (FWPCA, 1968).

Amphibians

The LD_{50} for bullfrogs was $>$2,000 mg/kg to abate when the animals were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Arthropods

The 48-hour LC_{50} for stoneflies (*Pteronarcys californica*), and amphipods (*Gammarus lacustris*) exposed to abate was 100 ppb and 1,500 ppb, respectively (FWPCA, 1968).

The 96-hour LC_{50} for the amphipod (*G. lacustris*) exposed to abate was 960 ppb (Sanders, 1969).

ALDRIN

Mammals

The LD_{50} for rats was 54 to 56 mg/kg and for rabbits, < 150 mg/kg to aldrin when the mammals were fed the stated dosages orally (Spector, 1955).

The frequency of estrus in rats decreased significantly when they were fed 10 or 20 ppm of aldrin from one month of age (Ball, Kay and Sinclair, 1953).

Birds

Tucker and Crabtree (1970) reported the LD_{50} for young mallards was 520 mg/kg; for young pheasants, 16.8 mg/kg; for young bobwhite quail, 6.6 mg/kg; and for fulvous tree duck, 29.2 mg/kg to aldrin when the birds were given the stated dosages orally in a capsule. The LC_{50} for mallards was 150 to 170 ppm; for pheasants was 50 to 60 ppm; and for coturnix, 30 to 40 ppm of aldrin in diets of 2-week-old birds fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a). The LC_{50} for bobwhite quail chicks was 39 ppm and for mallard ducklings, 164 ppm to aldrin when the birds were fed the stated dosages for 5 days and then clean food for 3 days (Heath and Stickel, 1965).

Aldrin in acetone injected into hen eggs at 100 ppm, 200 ppm, 300 ppm, and 400 ppm killed 30, 35, 48, and 70 percent of the embryos (Dunachie and Fletcher, 1969). Starvation experiments with chicks from eggs which had received 5 ppm showed complete mortality by the 5th day, whereas in the control mortality was about 50 percent.

Aldrin at 0.25 ppm in the diet of turkeys resulted in a highly significant growth depression in both sexes (Anderson, Blakely and MacGregor, 1961).

Wood pigeons in England were fed aldrin (40, 46, 53, 61, 70, and 80 mg/kg) under controlled laboratory conditions, and the toxicities and residues in flesh and various organs were measured (Turtle et al., 1963). After this investigation birds in the field were examined. The results of the laboratory and field analyses support the conclusion that aldrin was one of the main causes for wood-pigeon deaths in nature and prompted the Ministry of Agriculture, Fisheries and Food to discontinue the recommended use of aldrin as a seed dressing.

Fishes

See table 1 for the LC_{50} for various fish to aldrin.

The 96-hr LC_{50} for rainbow trout exposed to aldrin at temperatures of 1.6°C, 7.2°C, and 12.7°C was 24 ppb, 8.1 ppb, and 6.1 ppb, respectively (Macek, Hutchinson and Cope, 1969); and the 96-hour LC_{50} for bluegills exposed at temperatures of 12.7°C, 18.3°C, and 23.8°C was 39 ppb, 16 ppb, and 10 ppb, respectively. As both temperature and exposure time increased, the LC_{50} to small (about 1 g) bluegills decreased (table 2).

About 5 percent of the mosquito fish that survived an exposure to aldrin above the threshold toxicity of the insecticide aborted their young (Boyd, 1964).

TABLE 1. The LC_{50} for various fish to aldrin.

Fish Species	Exposure (Hours)	LC_{50} (ppm)	Source
Rainbow trout	24	0.036	Cope, 1965
Rainbow trout	24	0.03	Mayhew, 1955
Bluegill	24	0.096	Cope, 1965
Rainbow trout	48	0.003	FWPCA, 1968
Bluegill	96	0.015	Henderson, Pickering and Tarzwell, 1959
Goldfish	96	0.028	"

TABLE 2. Effects of increasing temperature and exposure time on the toxicity of aldrin to bluegills (Cope, 1965).

Temperature °F	LC_{50} (ppm)		
	24 hrs	48 hrs	96 hrs
45	0.130	0.0264	0.0097
55	0.0308	0.0135	0.0077
65	0.0164	0.0085	0.0062
75	0.0093	0.0067	0.0056

The results of a study by Moye and Luckmann (1964) indicated that a single application of aldrin at 2 lb/A for insect control in Milford, Illinois, in 1960 killed a large number of fish in a small exposed stream (see *Arthropods and Annelids*). A collection of fish 7 months later, however, showed the usual diversity of species and size of fish. Hence there appeared to be a rapid recovery of the fish population.

Three species of fish were collected in the field at Twin Bayou, Mississippi, where the population had been exposed to heavy concentrations of several insecticides used in the adjoining cotton acreages (Ferguson et al., 1965b). The toxicity to aldrin in these field-collected fish compared with a control population, as measured by 36-hour LC_{50}, were: golden shiner, control 80 ppb versus Twin Bayou 4,750 ppb; bluegills, control 38 ppb versus Twin Bayou 3,000 ppb; and green sunfish, control 62 ppb versus Twin Bayou 3,250 ppb. In another investigation resistant mosquito fish and black bullheads were collected from streams in Mississippi (Ferguson et al., 1965a). The toxicity to aldrin in these fish compared with an unselected control population, as measured by 36-hour LC_{50}, were: mosquito fish, control 50 ppb versus resistant (Sidon, Miss.) 2,100 ppb; and black bullhead, control 12.5 ppb versus resistant (Wayside, Miss.) 150 ppb.

Mosquito fish resistant to aldrin were also found in Mississippi Delta region (Ferguson, 1969). Cross-resistance was suggested as an explanation, as aldrin had not been used in the area during the preceding 8 years.

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles exposed to aldrin was 2.0 ppm (Sanders, 1970).

Mollusca

Only 1 ppb of aldrin in the water limited the development of clam eggs by 70 percent, and 1 ppm aldrin reduced the growth of mature oysters by 96 percent after a 7-day exposure (USDI, 1960).

Arthropods and Annelids

See table 8 for the LC_{50} for various arthropods to aldrin.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to aldrin was 23 ppb and 28 ppb, respectively (Sanders and Cope, 1966).

Populations of the mite *Tetranychus bimaculatus* on beans and potatoes increased up to 5-fold after the application of aldrin at dosages from 3 to 60 lb/A (Klostermeyer and Rasmussen, 1953).

The effects of aldrin on a small stream were studied in 1960, when approximately 23,000 acres of farmland near Milford, Illinois, were treated with aldrin at a dosage of 2 lb/A (Moye and Luckmann, 1964). A 6-mile-long segment of Sugar Creek flowing through the area was subjected to contamination during aerial application of the aldrin granules. The stream was sampled for some species of Ephemeroptera, Trichoptera, Elmidae (Coleoptera), and Chironomidae (Diptera) up to 19 months after treatment. Of the 4 aquatic taxa studied, only Elmidae appeared unaffected by the treatment. Species of Ephemeroptera decreased severely. Trichoptera and Chironomidae increased during the summer after treatment, but by the second spring after treatment populations of these taxa were at similar densities in the treated and untreated portions of Sugar Creek. The results of the study indicated that although a single application of aldrin severely reduced the number of Ephemeroptera, recovery was rapid, and no permanent damage resulted.

TABLE 8. The LC_{50} for various arthropods to aldrin

Arthropod Specie	[illegible]	LC_{50} (ppm)	Source
Sand shrimp	24	0.03	Eisler, 1969
Hermit crab	24	0.3	"
Grass shrimp	24	>9	"
Amphipod (*Gammarus lacustris*)	24	48	Sanders, 1969
Stonefly (*Pteronarcys californica*)	48	0.008	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	0.028	"
Amphipod (*G. lacustris*)	48	12	"

Polivka (1953 in Davey, 1963) reported that in Ohio aldrin applied at 5 lb/A significantly reduced the number of earthworms on a golf course.

Aldrin applied at 800 lb/A of 1.25 percent dust to year-old fallow plots did not affect the Lumbricidae, Enchytraeidae, or Nematoda (Edwards, Dennis and Empson, 1967), but significantly reduced the entomobryid or isotomid Collembola and Pauropoda. Both Coleoptera and Diptera biomass decreased. In general, the aldrin killed more pest species than predaceous or beneficial species.

Plants

The exposure of natural phytoplankton to 1 ppm of aldrin for 4 hours reduced its productivity by 84.6 percent (Butler, 1965a).

Aldrin applied to soils at 1, 2, or 3 lb/A (5-in. layer) was found to be translocated into alfalfa growing in the treated soil, and the same was observed for cucumbers growing in soil receiving 5 or 25 lb/A (Lichtenstein et al., 1965).

Seeds with a high oil content, such as soybeans and peanuts, had nearly 10 times as much aldrin residue as corn, which has less oil in its seeds (Bruce, Decker and Wilson, 1966).

Biological Concentration

When 4 species of algae (*Microcystis aeruginosa*, *Anabaena cylindrica*, *Scenedesmus quadricauda*, and *Oedogonium* sp.) were exposed to 1 ppm of aldrin for 7 days, they concentrated the toxicant into their protoplasm about 100-fold (Vance and Drummond, 1969).

Missouri cornfields treated with aldrin at 1.0 lb/A for at least 15 of the last 17 years indicated soil residues (primarily dieldrin, the degradation product of aldrin) of 0.31 ppm (Korschgen, 1970). Residues in various plants and animals were as follows: earthworms, 1.49 ppm; crickets, 0.23 ppm; *Harpalus* ground beetles, 1.10 ppm; *Poecilus* ground beetles, 9.67 ppm; white-footed mice, 0.08 ppm; toads, 3.56 ppm; garter snakes, 12.06 ppm; and corn, foxtail, and sunflower seeds, less than 0.02 ppm each. Exceptionally high residues (37.48 ppm) were recorded in *Poecilus* beetles during June 1967, and the author attributed this to "abnormally high soil moisture and predaceous feeding habits of these insects."

Microorganisms

Jones (1956) reported that aldrin from 0.01 to 1 percent in soil was considerably more toxic to bacteria converting ammonia to nitrates in the soil than to bacteria changing organic matter to ammonia.

Persistence

When applied at a rate of 100 ppm to sandy loam soil, the remaining aldrin after 14 years was 40 percent (Nash and Woolson, 1967), and aldrin applied at 25 ppm to soil persisted (50-percent loss) for >4 years.

ALLETHRIN

Mammals

The LD_{50} for rats was 920 mg/kg to allethrin when the mammals were fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Birds

The LD_{50} for young mallards was >2,000 mg/kg to allethrin when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The 24-hour LC_{50} for rainbow trout exposed to allethrin was 80 ppb (Cope, 1965); and the 48-hour LC_{50} for rainbow trout was reported as 19 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for stoneflies (*Pteronarcys californica*), waterfleas (*Daphnia pulex*), and amphipods (*Gammarus lacustris*) exposed to allethrin was 28 ppb, 21 ppb, and 90 ppb, respectively (FWPCA, 1968).

The 96-hour LC_{50} for amphipod (*G. lacustris*) exposed to allethrin was 38 ppb (Sanders, 1969).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *D. pulex*, to allethrin was 56 ppb and 21 ppb, respectively (Sanders and Cope, 1966).

AMINOCARB

Mammals

The LD_{50} for rats was 50 mg/kg (FCH, 1970) and for mule deer 7.5 to 15 mg/kg (Tucker and Crabtree, 1970) to aminocarb when the mammals were given the stated dosages orally in a capsule.

Birds

The LD_{50} for young mallards was 22.5 mg/kg and for young pheasants, 42.4 mg/kg to aminocarb when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Arthropods

The 96-hour LC_{50} for amphipod (*Gammarus lacustris*) exposed to aminocarb was 39 ppb (Sanders, 1969).

ARAMITE

Mammals

The LD_{50} for rats was 3,900 mg/kg to aramite when the mammals were fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 48-hour LC_{50} for bluegill exposed to aramite was 35 ppb (FWPCA, 1968).

Arthropods and Annelids

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) and amphipods (*Gammarus lacustris*) exposed to aramite was 345 ppb and 100 ppb, respectively (FWPCA, 1968).

The 24-hour LC_{50} for amphipod (*G. lacustris*) exposed to aramite was 360 ppb (Sanders, 1969).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *D. pulex*, to aramite was 180 ppb and 160 ppb, respectively (Sanders and Cope, 1966).

Aramite applied as a wettable powder at 0.036 percent in 100 gallons of water had little or no effect on the earthworm (*Caloglyphus anomalus*) (Hyche, 1956).

AROCHLORS

Mammals

The LD_{50} for rats was estimated at 250 mg/kg to Arochlor when the mammals were fed the stated dosage orally (Bennett, Drinker and Warren, 1938); guinea pigs were estimated to require 170 mg/kg (Miller, 1944).

Guinea pigs, rats, and rabbits were given Arochlor at doses ranging from 17 to 1,380 mg as either single or multiple doses by subcutaneous injections (Miller, 1944). The 2 most common findings were liver damage and skin changes(similar to chloracne in man). Liver damage in rats from Arochlor was also reported by Bennett, Drinker and Warren (1938) through exposures by feeding and inhalation.

Birds

Phenochlor DP6 fed to coturnix at a dosage of 2,000 ppm in their diet killed all (n=20) the birds within 55 days (Koeman, Ten Noever de Brauw and de Vos, 1969).

Arochlor 1242, 1254, 1260, and 1268 fed to mallard ducks at a dosage of 2,000 mg/kg were not fatal (Tucker and Crabtree, 1970).

Regular egg-laying turned to scattered production and finally stopped for a week when coturnix were fed a single oral dose of 500 mg/kg (Arochlor 1254) (Tucker, unpublished, in Peakall and Lincer, 1970). The scattered eggs were found to be 9 percent thinner than normal, but returned to control level when the Arochlor effect wore off. Mallards fed 1,000 mg/kg of Arochlor produced 1 or no eggs before ceasing all production for 1 to 2 weeks. The eggs produced had shells 18 percent thinner, but again were normal when full production was resumed.

Ten-day-old mallard ducklings fed Arochlor at concentrations of 25, 50, and 100 ppm in their diet suffered no apparent effects (Friend and Trainer, 1970). However, when these ducklings were challenged with duck hepatitus virus 5 days later, they suffered significantly higher mortality (increased from 14 percent to a range of 39 to 68 percent) than the ducklings which did not receive Arochlor in their diet.

The LC_{50} to Arochlor (1232, 1242, 1248, 1254, 1260, and 1262) expressed as ppm in dry feed for 2-week-old penned mallards, pheasants, bobwhites, and coturnix fed the treated diets for 5 days is shown in table 4. Low levels of Arochlor 1254 (25 and 50 ppm) produced no measurable effects in mallards and bobwhite (Heath et al., 1970b).

TABLE 4. Toxicity (LC_{50}) of Arochlor in diets of 2-week-old birds (Heath et al., 1970b).

Formulation	Mallard	Pheasant	Bobwhite	Coturnix
1232	....	3150	3000	>5000
1242	3180	2080	2100	>5000
1248	2795	1310	1175	4845
1254	2700	1090	605	2900
1260	1975	1260	745	2185
1262	3010	1235	870	3000

AZINPHOS-METHYL

Mammals

The LD_{50} for the rat was 16 mg/kg to azinphos-methyl when the mammal was fed the stated dosage orally (Metcalf, Flint and Metcalf, 1962).

Birds

Tucker and Crabtree (1970) computed the LD_{50} for young mallards as 136 mg/kg; for young pheasants, 74.9 mg/kg; and for young chukar partridges, 84.2 mg/kg to azinphos-methyl when the birds were fed the stated dosages orally in capsules. The LC_{50} for mallards was 1,000 to 2,000 ppm; for pheasants, 1,000 to 2,000 ppm; for bobwhites, 400 to 500 ppm; and for coturnix, 600 to 700 ppm of azinphos-methyl in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

When chickens were fed azinphos-methyl at a dosage of 40 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The LC_{50} for various fish to azinphos-methyl is found in table 5.

TABLE 5. The LC_{50} for various fish to azinphos-methyl.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Rainbow trout	24	0.040	Cope, 1965
Harlequin fish	24	0.12	Alabaster, 1969
Brown trout	96	0.004	Macek and McAllister, 1970
Largemouth bass	96	0.005	"
Yellow perch	96	0.013	"
Rainbow trout	96	0.014	"
Bluegill	96	0.022	"
Redear sunfish	96	0.052	"
Coho salmon	96	0.017	"
Fathead minnow	96	0.235	"
Carp	96	0.695	"
Channel catfish	96	3.290	"
Black bullhead	96	3.500	"
Goldfish	96	4.270	"

The relative toxicity of azinphos-methyl to 2 species of fishes, as measured by the 48-hour EC_{50}, was as follows: rainbow trout at 80 ppb, 18°C, and bluegill at 2 ppb, 24°C (Cope, 1966).

The 24-hour LC_{50} for rainbow trout exposed to azinphos-methyl at temperatures of 1.6°C, 7.2°C, and 12.7°C was 26 ppb, 15 ppb, and 13 ppb, respectively (Macek, Hutchinson and Cope, 1969); and the 24-hour LC_{50} for bluegills exposed at temperatures of 12.7°C, 18.3°C, and 23.8°C was 16 ppb, 16 ppb, and 16 ppb, respectively.

In tests the 96-hour LC_{50} of azinphos-methyl was 0.2 ppm for carp, 0.04 for tilapia, and 0.008 ppm for mullet (Lahav and Sarig, 1969); the highest nonlethal dosage was 0.1 ppm for carp, 0.008 ppm for tilapia, and 0.004 ppm for mullet.

An investigation of the persistence of azinphos-methyl in fish revealed that 50 percent of the chemical was lost in <1 week (Meyer, 1965).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles exposed to azinphos-methyl was 0.66 ppm (Sanders, 1970).

Arthropods and Annelids

The LC_{50} for various arthropods to azinphos-methyl is found in table 6.

The 48-hour EC_{50} (immobilization value at 60°

TABLE 6. The LC_{50} for various arthropods to azinphos-methyl

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	0.00056	Sanders, 1969
Stonefly (*Pteronarcys californica*)	24	0.022	Sanders and Cope, 1968
Waterflea (*Daphnia magna*)	48	0.0002	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.0005	"
Waterflea (*D. pulex*)	48	0.0032	Sanders and Cope, 1966
Stonefly (*P. californica*)	48	0.008	"
" (*P. californica*)	48	0.008	FWPCA, 1968

$_{50}$) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to azinphos-methyl (ethyl guthion) was 4.2 ppb and 3.2 ppb, respectively (Sanders and Cope, 1966).

The toxicity of azinphos-methyl to 3 species of invertebrates, as measured by the 48-hour EC$_{50}$, was as follows: stonefly nymph (*Pteronarcys californicus* [*sic*]) at 8 ppb, waterflea (*S. serrulatus*) at 4 ppb, and waterflea (*D. pulex*) at 3 ppb (Cope, 1966).

Hopkins and Kirk (1957) reported the 96-hour LD$_{50}$ for azinphos-methyl tested against earthworms (*Eisenia* sp.) as 12.2 lb/A.

In greenhouse tests alfalfa treated with azinphos-methyl at 0.8 lb/A after 9 days' exposure caused about a 90-percent mortality in pollinating leafcutting bees (Waller, 1962).

BINAPACRYL

Mammals

The LD$_{50}$ for the rat was 120 to 165 mg/kg to binapacryl when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

Binapacryl in acetone injected into hen eggs at 1 ppm, 5 ppm, 10 ppm, 25 ppm, 50 ppm, and 100 ppm killed 53, 78, 89, 100, 95, and 100 percent of the embryos (Dunachie and Fletcher, 1969).

Arthropods

Van de Vrie (1962) reported that binapacryl at concentrations of 0.10 and 0.05 percent in 2 days would kill 94 and 86 percent of predatory mites (*Typhlodromus tiliae* and *T. tiliarum*). In later studies Van de Vrie (1967) reported that binapacryl at 0.10-percent concentration applied to apple trees was harmless to the predatory bug *Anthocoris nemorum*, but caused some mortality to another predatory bug, *Orius* sp.; this concentration did cause a heavy mortality to the woolly-aphid parasite population (*Aphelinus mali*).

BROMOPHOS

Mammals

The LD$_{50}$ for the rat was 3,750 to 8,100 mg/kg to bromophos when the mammals were fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC$_{50}$ for harlequin fish to bromophos was 1.9 ppm (Alabaster, 1969).

CARBARYL

Mammals

The LD$_{50}$ for the rat was 540 mg/kg (Metcalf, Flint and Metcalf, 1962) and for mule deer, 200 to 400 mg/kg (Tucker and Crabtree, 1970) to carbaryl when the mammals were given the stated dosages orally in a capsule.

Cotton rat reproduction was delayed by carbaryl application (2 lb/A) to a grassland, and this resulted in a reduced population (Barrett, 1968). In laboratory tests carbaryl was fed to cotton rats orally at 1.1 mg/day per individual for 10 days. In these rats weighing from 140 to 180 g both the number of litters born and number of females giving birth were reduced by more than 50 percent. There appeared to be, however, no effect on either the mouse (house) or the old-field mouse populations by the carbaryl (2 lb/A) application.

Birds

The LD$_{50}$ for young mallards was $\geq$2,179 mg/kg; for young pheasants, $\geq$2,000 mg/kg; for young coturnix, 2,290 mg/kg; for pigeons (*Columba livia*), 1,000 to 3,000 mg/kg; for sharp-tailed grouse, 780 to 1,790 mg/kg; and for Canada geese, 1,790 mg/kg to carbaryl when the birds were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970). The LC$_{50}$ for mallards was $\geq$5,000 ppm; for pheasants, $\geq$5,000; for bobwhites, $\geq$5,000 ppm; and for coturnix, $\geq$5,000 ppm of carbaryl in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

Carbaryl in acetone injected into hen eggs at 100 ppm and 200 ppm killed 61 and 100 percent of the embryos (Dunachie and Fletcher, 1969). This toxicant also caused teratogenic effects at 50 ppm and above.

When chickens were fed carbaryl at a dosage of 1,600 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action unknown.

Fishes

The LC_{50} for various fish to carbaryl is found in table 7.

The toxicity of carbaryl to 3 species of fish, as measured by the 48-hour EC_{50}, was as follows: channel catfish at 10,000 ppb, 24°C; bluegill at 6,800 ppb, 24°C; and rainbow trout at 2,000 ppb, 18°C (Cope, 1966).

TABLE 7. The LC_{50} for various fish to carbaryl.

Fish Species	Exposure (hr)	LC_{50} (ppm)	Source
Longnose killifish	24	1.75	Stewart, Milleran and Breese, 1967
Harlequin fish	24	3.4	Alabaster, 1969
Shiner perch	24	3.9	Stewart, Milleran and Breese, 1967
English sole	24	4.1	"
White mullet	24	4.25	"
Three-spine stickleback	24	6.7	"
Brown trout	48	1.5	FWPCA, 1968
Yellow perch	96	0.745	Macek and McAllister, 1970
Coho salmon	96	0.764	"
Brown trout	96	1.95	"
Rainbow trout	96	4.38	"
Carp	96	5.28	"
Largemouth bass	96	6.4	"
Bluegill	96	6.76	"
Redear sunfish	96	11.2	"
Fathead minnow	96	13.0	Stewart, Milleran and Breese, 1967
Goldfish	96	13.2	Macek and McAllister, 1970
Fathead minnow	96	14.6	"
Channel catfish	96	15.8	"
Black bullhead	96	20.0	"

In laboratory experiments conducted by Mr. Jack Lowe, fish were exposed to carbaryl and 2,4-D (no dosage given) for 1 to 5 months (Butler, 1969a). The exposed fish grew as well as the controls and had little mortality; however, careful examinations revealed massive invasions of the nervous system of the test fish by what appeared to be a microsporidian parasite. The author suggested that the pesticides lowered the natural resistance of the fish to parasite attack.

Mollusca

As little as 1 ppb of carbaryl was found to inhibit the development of clam eggs (USDI, 1960). The EC_{50} for carbaryl tested against various species of fish for different exposure times was as follows: bay mussel, 2.3 ppm, 48 hours; Pacific oyster, 2.2 ppm, hours; and cockel clam, 7.3 ppm, 24 hours (Stewart, Milleran and Breese, 1967).

Arthropods and Annelids

The LC_{50} for various arthropods to carbaryl is found in table 8.

Brown shrimp tolerated carbaryl at 27 ppb, whereas the tolerance for white shrimp was only 15 ppb (USDI, 1960).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to carbaryl was 7.6 ppb and 6.4 ppb, respectively (Sanders and Cope, 1966).

A suspension of 0.1 percent of carbaryl was found to be "extremely toxic" to earthworms (An der Lan and Aspock, 1962).

An experiment testing the toxicity of carbaryl to honeybees showed the insecticide to be highly toxic (Morse, 1961). Mortalities were above normal for up to 3 weeks after insecticide application, and within 47 days after treatment the treated colonies had lost more than 6 times as many bees as the untreated colonies.

In greenhouse tests alfalfa treated with carbaryl at 1 lb/A after 10 days' exposure caused only about a 20-percent mortality in the pollinating leafcutting bee (Waller, 1969).

TABLE 8. The LC_{50} for various arthropods to carbaryl

Arthropod Species	Exposure (hours)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcella badia*)	24	0.005	Sanders and Cope, 1968
" (*Claassenia sabulosa*)	24	0.013	"
" (*Pteronarcys californica*)	24	0.030	"
Amphipod (*Gammarus lacustris*)	24	0.040	Sanders, 1969
Mud shrimp	24	0.04-0.13	Stewart, Millemann and Breese, 1967
Ghost shrimp	24	0.13	"
Shore crab	24	0.27-0.71	"
Dungeness crab	24	0.60-0.88	"
Stonefly (*P. californica*)	48	0.0013	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	0.006	Cope, 1966
" (*D. pulex*)	48	0.0064	FWPCA, 1968
" (*Simocephalus serrulatus*)	48	0.008	Cope, 1966
Stonefly (*P. californica*)	48	0.018	"
Amphipod (*G. lacustris*)	48	0.025	FWPCA, 1968
Ghost shrimp	48	0.03-0.08	Stewart, Millemann and Breese, 1967
Red crawfish	48	8.6	Muncy and Oliver, 1963

Carbaryl at a concentration of 1 ppm did not prevent egg hatching of the Dungeness crab, but prevented moulting of all prezoeae to zoeae (Buchanan, Millemann and Stewart, 1970). Moulting was delayed at 0.0001 ppm. The 96-hour LC_{50} for first stage zoeae was 0.01 ppm of carbaryl. Survival of zoeae after a 25-day exposure to concentrations of 0.0001, 0.00032, 0.001, 0.0032, and 0.01 ppm were 88, 60, 69, 21, and 0 percent, respectively. Adult crabs were paralyzed (32 percent) within 6 hours after they had fed on cockle clams which had been exposed for 24 hours to 1 ppm of carbaryl.

Both biomass and numbers of arthropods were reduced by more than 95 percent in a carbaryl-treated (2 lb/A) area (Barrett, 1968). The arthropod number remained well below the numbers in the untreated area for 5 weeks, but after 7 weeks total biomass at least had returned to normal. Phytophagous insects (both Homoptera and Hemiptera) were more severely affected than predaceous insects and spiders. The spiders were back to normal density within 3 weeks after treatment.

Plants

No effect of carbaryl (2 lb/A) was detected on plants (primarily millet) (Barrett, 1968); however, carbaryl was toxic to algae at concentrations above 0.1 ppm and reduced their growth (Christie, 1969).

Microorganisms

Percentage litter decomposition in a grassland treated with carbaryl (2 lb/A) was only 21 percent, compared with 28 percent in the untreated control 8 weeks after spraying (Barrett, 1968).

Persistence

Carbaryl applied at a rate of 2 lb/A resulted in residues of 35 ppm on the plants, but these residues decreased rapidly and by the 16th day after application the residue was only 0.87 ppm (Barrett, 1968).

CARBOFURAN

Mammals

The LD_{50} for the rat was 11 mg/kg to carbofuran when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Birds

The LD_{50} for young mallards was 0.40 mg/kg; for young pheasants, 4.2 mg/kg; for young bobwhite quail, 5.0 mg/kg; and for young fulvous ducks, 0.94 mg/kg to carbofuran when the birds were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970).

CARBOPHENOTHION

Mammals

The LD_{50} for male rats was 53.9 mg/kg to carbophenothion when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

When chickens were fed carbophenothion (methyl) and carbophenothion at dosages of 890 and 640 mg/kg respectively, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The 48-hour LC_{50} for bluegill exposed to carbophenothion was 225 ppb (FWPCA, 1968).

The 24-hour LC_{50} for harlequin fish to carbophenothion was 3.4 ppm (Alabaster, 1969).

Amphibians

The 24-hour LC_{50} for chorus frog tadpoles exposed to carbophenothion was 0.10 ppm (Sanders, 1970).

Arthropods

The 48-hour LC_{50} for waterfleas (*Simocephalus serrulatus*) and amphipods (*Gammarus lacustris*) exposed to carbophenothion was 0.009 ppb and 28 ppb, respectively (FWPCA, 1968).

Persistence

Carbophenothion applied to soil persisted for >6 months (Mulla, Georghiou and Cramer, 1961).

CHLORBENSIDE

Birds

Chlorbenside in acetone injected into hen eggs at up to 500 ppm caused little or no mortality to the embryos (Dunachie and Fletcher, 1969).

CHLORDANE

Mammals

The LD_{50} for rats was 200 to 590 mg/kg; for mice, 430 mg/kg; and for rabbits, 100 to 300 mg/kg to chlordane when the mammals were fed the stated dosages orally (Spector, 1955).

Birds

The LD_{50} for young mallards was 1,200 mg/kg to chlordane when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was 800 to 880 ppm; for pheasants, 400 to 500 ppm; and for coturnix, 300 to 380 ppm of chlordane in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a). The LC_{50} for bobwhite quail chicks to chlordane was 330 ppm when the birds were fed the stated dosage for 5 days and then fed clean food for 3 days (Heath and Stickel, 1965).

Chlordane in acetone injected into hen eggs at up to 500 ppm caused no mortality to the embryos (Dunachie and Fletcher, 1969).

When a marsh in North Dakota was treated with chlordane at a rate of 1 lb/A, neither the blue-winged teal nor the shoveller produced any young, and the number produced by the coot and red-wing blackbird was reduced by about 60 percent (Hanson, 1952). Much of the effect was believed due to the nearly complete destruction of insect life on which these birds feed.

Fishes

The LC_{50} for various fish to chlordane is found in table 9.

TABLE 9. The LC_{50} for various fish to chlordane

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Rainbow trout	24	0.022	Cope, 1965
Rainbow trout	24	0.05	Henderson, Pickering and Tarzwell, 1959
Bluegill	24	0.056	Cope, 1965
Rainbow trout	48	0.010	FWPCA, 1968
Bluegill	96	0.022	Henderson, Pickering and Tarzwell, 1959
Goldfish	96	0.082	"

The 24-hour LC_{50} for bluegills exposed to chlordane at temperatures of 12.7°C, 18.3°C, and 23.9°C was 220 ppb, 170 ppb, and 95 ppb, respectively (Macek, Hutchinson and Cope, 1969).

A natural population of mosquito fish in ditches adjacent to cotton fields was found to be 20 times more resistant to chlordane than normal (Boyd and Ferguson, 1964b).

Arthropods and Annelids

The LC_{50} for various arthropods to chlordane is found in table 10.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to chlordane was 20 ppb and 29 ppb, respectively (Sanders and Cope, 1966).

Mite populations (*Tetranychus bimaculatus*) on beans and potatoes increased up to 2 times after applications of chlordane made at 8 to 75 lb/A (Klostermeyer and Rasmussen, 1953).

Chlordane applied at rates of 5, 10, and 20 lb/A to a golf course in Ohio caused a significant reduction in earthworm populations, measured one year after treatment (Polivka, 1953 in Davey, 1963).

Chlordane at 10 lb/A in another test eliminated earthworms in the treated plots (Doane, 1962). In studies in Germany exposure of *Lumbricus rubellus* to chlordane as a 0.25-percent emulsion or 1- to 5-percent dust in the laboratory caused significant mortalities in the earthworms (Van der Drift, 1963). Also in England, chlordane applied at recommended dosages for the control of soil insect pests destroyed the earthworms present (Raw, 1963).

TABLE 10. The LC_{50} for various arthropods to chlordane

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	96	0.160	Sanders, 1969
Stonefly (*Pteronarcys californica*)	96	0.170	Sanders and Cope, 1968
Waterflea (*Simocephalus serrulatus*)	48	0.020	FWPCA, 1968
Stonefly (*P. californica*)	48	0.055	"
Amphipod (*G. lacustris*)	48	0.080	"

Plants

Chlordane at 1 ppm for a 4-hour exposure period caused a 94-percent reduction in the productivity of natural phytoplankton communities in the laboratory (Butler, 1963a).

Chlordane at 1, 10, and 100 ppm in soil caused significant changes in the macro and micro element (N, P, K, Ca, Mg, Mn, Fe, Cu, B, Al, Sr, and Zn) constituents of above-ground portions of corn and bean plants (Cole et al., 1968). For example, iron content in beans was higher (401 ppm) in the chlordane (10 ppm) treated than in the control (252 ppm) at the end of 4 weeks of growth; however, the aluminum content was lower (82 ppm) in the chlordane (10 ppm) treated than the control (217 ppm) at the end of 8 weeks of growth.

Microorganisms

Chlordane at 0.01 to 1 percent in soil has been shown to be considerably more toxic to bacteria converting ammonia to nitrates in soil than those changing organic matter to ammonia (Jones, 1956).

Biological Concentration

Eastern oysters, exposed in flowing seawater to chlordane at 0.01 ppm in the laboratory, concentrated the toxicant within 10 days to a level of 7,300 times the ambient concentration (Wilson, 1965).

Persistence

Chlordane applied at 25 lb/A persisted in soil for ≥ 12 years (Lichtenstein and Polivka, 1959). Nash and Woolson (1967) found that chlordane applied at 50 ppm to soil persisted (50 percent loss) for 6 years, and when applied at 100 ppm to sandy loam soil persisted (60 percent loss) for 14 years.

CHLORDECONE

Birds

Hens fed chlordecone at levels of 75 ppm and 150 ppm in their diet for 12 weeks produced significantly ($P<0.05$) fewer eggs (Naber and Ware, 1965). Hens at the higher rate lost weight, and chicks from these hens exhibited a nervous syndrome. Chick survival was also reduced.

Fishes

The 48-hour LC_{50} for rainbow trout exposed to chlordecone was 87.5 ppb (FWPCA, 1968).

CHLORFENVINPHOS

Mammals

The LD_{50} for the rat was 10 to 39 mg/kg to chlorfenvinphos when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to chlorfenvinphos was 0.85 ppm (Alabaster, 1969).

CHLOROBENZILATE

Mammals

The LD_{50} for rats was 960 mg/kg to chlorobenzilate when the mammals were fed the stated dosage orally (FCH, 1970).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to chlorobenzilate was 710 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for waterfleas (*Simocephalus serrulatus*) exposed to chlorobenzilate was 550 ppb (FWPCA, 1968).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *S. serrulatus* and *Daphnia pulex*, to chlorobenzilate was 550 ppb and 870 ppb, respectively (Sanders and Cope, 1966).

CHLOROPROPYLATE

Mammals

The LD_{50} for the rat was 5,000 mg/kg to chloropropylate when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to chloropropylate was 22 ppm (Alabaster, 1969).

CHLOROTHION

Mammals

The LD_{50} for male rats was 880 mg/kg to chlorothion when the mammals were fed the stated dosage orally (FCH, 1970).

Arthropods

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) exposed to chlorothion was 4.5 ppb (FWPCA, 1968).

CIODRIN

Mammals

The LD_{50} for rats was 125 mg/kg to Ciodrin when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

When chickens were fed Ciodrin at a dosage of 100 mg/kg, the chickens developed leg weakness (Gaines, 1969). The mode of action was unknown.

Arthropods

The 96-hour LC_{50} for the amphipod (*Gammarus lacustris*) exposed to Ciodrin was 46 ppb (Sanders, 1969).

COUMAPHOS

Mammals

The LD_{50} for the rat was 56 to 230 mg/kg to coumaphos when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for young mallards was 29.8 mg/kg to coumaphos when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for pheasants was 300 to 400 ppm and for coturnix, 200 to 250 ppm to coumaphos in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

When chickens were fed coumaphos at a dosage of 100 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The 24-hour LC_{50} for harlequin fish to coumaphos was 0.082 ppm (Alabaster, 1969).

Arthropods

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) and amphipods (*Gammarus lacustris*) exposed to coumaphos was 1 ppb and 0.14 ppb, respectively (FWPCA, 1968).

The 96-hour LC_{50} for the amphipod (*G. lacustris*) exposed to coumaphos was 0.88 ppb (Sanders, 1969).

CRUFOMATE

Mammals

The LD_{50} for female rats was 770 mg/kg to crufomate when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

When chickens were fed crufomate at a dosage of 400 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

CRYOLITE

Mammals

The LD_{50} for rats was >10,000 mg/kg to cryolite when the mammals were fed the stated dosage orally (FCH, 1970).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to cryolite was 47,000 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for waterfleas (*Daphnia pulex*) exposed to cryolite was 5 ppm (FWPCA, 1968).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *D. pulex*, to cryolite was 10 ppm and 5 ppm, respectively (Sanders and Cope, 1966).

DDT

Mammals

The LD_{50} for the rat was 420 to 800 mg/kg; for the mouse, 300 mg/kg; for the rabbit, 250 to 400 mg/kg; for the dog, 60 to 75 mg/kg; and for the guinea pig, 400 mg/kg to DDT when the mammals were fed the stated dosages orally (Spector, 1956).

Northern white-footed mouse populations were not noticeably affected by DDT applied at a rate of 2 lb/A to a forest in Maryland (Stickel, 1946 and 1951).

Populations of the white-footed mouse in New Jersey woods adjacent to treated crop fields were exposed to DDT at 0.12 to 0.21 lb/A and parathion at 0.01 to 0.06 lb/A (Jackson, 1952). Presumably because the level of contamination of the adjacent woods was low and the ingestion of insecticides was quite small, the white-footed mouse population was not measurably affected.

Fir and pine forest treatments with DDT at 1, 5, and 7½ lb/A for insect control caused no significant decrease in populations of coeur d'Alene chipmunks, buff-bellied chipmunks, sagebrush white-footed mice, redbacked mice, jumping mice, pine squirrels, Columbian ground squirrels, pocket gophers, black bears, and white-tailed deer (Adams et al., 1949). Only a few chipmunks appeared to be affected by the 7½-lb dosage of DDT.

Fish from the Miramichi River in New Brunswick which were naturally contaminated with DDT were incorporated in a feed mixture and fed to mink (Gilbert, 1969). These animals developed high levels of DDT in their livers and adipose tissue. Spleen and adrenal weights, erythrocyte and leukocyte counts, hemoglobin, and hematocrit were all influenced by the DDT in the ration. Females on the diet with DDT produced fewer young (4.8 kits) than did the control (5.9 kits). Embryonic loss (counting losses to 24 hours post birth) was significantly greater ($P<0.01$) in the DDT-exposed females.

A colony of mice was fed DDT and selected for resistance (Ozburn and Morrison, 1964). The LD_{50} for the selected colony after 10 generations was 900 mg/kg, whereas the LD_{50} for the control colony was 550 mg/kg. This experiment documented that mice, as well as insects, can evolve resistance to DDT.

Adult mice (house) fed diets containing 200 and 800 ppm of DDT were observed to have a higher death rate of females during the gestation period, in males, and in young produced (Cannon and Holcomb, 1968).

In an experiment by Hayne (1970), DDT was applied to forest at rates of ⅛, ¼, and 2 lb/A. He found that only about 19 percent of the spray reached the forest floor immediately. A year later the residues in both soil and litter had increased to about 99 percent of the amount applied, in part due to wash-off and leaf fall. No mortality was observed in the white-footed mouse population. DDT accumulated rapidly (7 to 10 days) in the fat of the animals. This was especially true of 2 lb/A dosage where the animals accumulated about 22 ppm of total DDE, DDT, and TDE in 7 days. The animals tended to lose their burden of DDT and 376 days after spraying had lost all additional DDT from the spraying operation.

Birds

Laboratory experiments. Tucker and Crabtree (1970) reported the LD_{50} for young mallards as $>2,240$ mg/kg; for young pheasants, 1,296 mg/kg; for young coturnix, 841 mg/kg; for pigeons (*Columba livia*), $>4,000$ mg/kg; and for lesser sandhill cranes, $>1,200$ mg/kg to DDT when the birds were given the stated dosages orally in a capsule.

The LC_{50} for mallards was 850 to 1,200 ppm; for pheasants, 300 to 700 ppm; for bobwhites, 600 to 1,000 ppm; and for coturnix, 400 to 800 ppm of DDT in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a). The LC_{50} for mallards was 3,300 to 3,800 ppm; for pheasants, 750 to 850 ppm; for bobwhites, 750 to 850 ppm; and for coturnix, 1,200 to 1,400 ppm of DDE (a metabolite of DDT) in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

The LD_{50} for 7-day-old pheasants to p,p'-DDT, p,p'-TDE, p,p'-DDE, and technical DDT in their diets was 550 ppm, 522 ppm, 1,086 ppm, and 955 ppm, respectively (Gill, Verts and Christensen, 1970). The LD_{50} to o,p'-DDT was not established, but was estimated to be in excess of 5,000 ppm.

Pheasants were maintained on diets containing different dosages of DDT for an experimental period of 90 days (Genelly and Rudd, 1956). In the test 8 out of the 10 females on 800 ppm of DDT died; all the 4 males on 400 ppm of DDT died, whereas the 20 females on this dosage all survived; and 1 out of the 10 females on 200 ppm of DDT died.

DDT in acetone injected into hen eggs at up to 500 ppm caused little or no mortality to the embryos (Dunachie and Fletcher, 1969). However, when chicks, hatched from eggs which had received 100 ppm of DDT, were starved for 4 days, all died. Untreated controls handled in a similar manner resulted in only about a 50-percent mortality.

DDT fed daily to pheasant hens at 10, 100, and 500 ppm DDT in their food produced a normal number of eggs which were fertile and hatched satisfactorily (Azevedo, Hunt and Woods, 1965); however, chick mortalities were reported to be highest among young from parents receiving 500 ppm of DDT.

DDT has been reported not only to be toxic to birds but also to cause significant changes in the physiology of some species of birds. In Bengalese finches, for example, DDT stimulated these birds to produce eggs with significantly heavier eggshells (Jefferies, 1969). Ovulation in these finches was also delayed (nearly twice normal) by the daily administration per individual of a dosage of 270 μg of DDT (Jefferies, 1967).

DDT residues experimentally produced in cowbirds caused death (35 to 99 ppm in brain) in these birds at about the same dosage of DDT as found in dead robins, sparrows, and eagles (17 to 188 ppm in brain) collected in nature (Stickel, Stickel and Christensen, 1966).

Only 44 percent of the eggs laid by herring gulls on the Lake Michigan side of the Door County peninsula were observed to hatch, as compared with a 90-percent level of hatching found in the same species in Denmark. This reduction was reportedly due to the higher level of DDT and its metabolites found in the Michigan gull eggs (Keith, 1966).

Bald eagles fed controlled dosages of DDT in the laboratory (Stickel et al., 1966) proved to be only moderately susceptible to DDT; about 80 ppm (dry weight) of DDT in the diet of the eagles was estimated to be the LD_{50} for this bird. The authors pointed out that this level produced chronic poisoning, and suppression of reproduction or eggshell thinning may take place at much lower dosages. Lethal residues in the brain ran between 58 and 88 ppm (wet weight), a level similar to that for other species. These results agree favorably with previous studies by Stickel, Stickel and Christensen (1966) when they found that residues of DDT in the brain between 45 and 100 ppm frequently induced death in both birds and mammals.

In a long-term study of DDT kinetics in cowbirds, about 50 birds were fed DDT 40 ppm in oil in their diets for 6 weeks with loss of only 2 birds (Stickel, 1965). All birds were fed clean food thereafter, yet 7 more birds died. All but one of these deaths occurred after the birds' exposure to unusual disturbances; typical DDT tremors were observed in several. One of these disturbances was 2 persons entering the cage to capture a few birds for residue analyses.

When white king pigeons were fed DDT (10 ppm) in their feed for 1 week steroid metabolism (testosterone increased from 35.7 to 75.4 mμ moles and progesterone increased from 30.1 to 78.8 mμ moles) was significantly increased (Peakall, 1967).

House sparrows and coturnix fed earthworms containing DDT of 298 ppm (wet weight) died within 1 to 10 days (Boykins, 1967). Sparrows fed earthworms containing 58 to 90 ppm (wet weight) of DDT survived 2 to 6 days. These levels of DDT in earthworms were relatively high; the highest level detected in earthworms on the Michigan State campus was 139 ppm of DDT.

In New Jersey from 1880 to 1938 the mean eggshell weight of 117 osprey eggshells was 7.08± 0.068 g, whereas in 1957 the mean weight of 6 eggshells was 5.30±0.440 g, indicating a decline of 25 percent. Associated with the decline in eggshell weight has been a decline in the osprey population in this region (Hickey and Anderson, 1968).

A total of 614 peregrine falcon eggshells were both weighed and measured for thickness (Hickey and Anderson, 1968). Eggs collected in California from 1947 to 1952 had a significant decrease (95 percent confidence) in both thickness and weight of eggshells, compared with those collected in the same area from 1895 to 1939. In the same study a regression analysis was run between shell thickness and total DDE residues in herring gull eggs collected in Rhode Island, Maine, Michigan, Minnesota, and Wisconsin. A high correlation ($P = 0.001$) was found between the level of DDE residue and the thickness of the eggshell: the more DDE, the thinner the eggshell.

The weights of raptor (bald eagle, osprey, peregrine falcon, and herring gull) eggshells in museum and private collections were measured to determine if there had been a change in the weights of these eggshells from the pre-DDT (1880 to 1939) to the post-DDT period (Hickey and Anderson, 1968). In Brevard County, Florida, bald eagle eggshells from the pre-DDT era weighed 12.15±0.127 g, 51 eggs measured; eggshells from 1947 to 1962 (post-DDT era) weighed 9.96±0.280 g, 19 eggs measured. Hence there was an 18-percent decrease in the weight of the eggshells.

Reports were also received that the population of the bald eagle was declining in this area. Similar results were reported from Osceola County, Florida; from 1901 to 1944, the mean weight for bald eagle eggshells was 12.34±0.240, whereas from 1950 to 1962 the mean weight was 9.88±0.140. In addition to a decline of 20 percent in the weight of bald eagle eggshells, the populations of the bald eagle were reported declining in this county.

Fyfe et al. (1969) in Canada reported a significant drop (11 percent) in the thickness of the prairie falcon eggs, compared with eggs sampled from the pre-organochlorine-insecticide era. Although other chemicals were present in addition to DDT, a high correlation was found between eggshell thickness and DDE residue in the eggs. Associated with the decline in eggshell thickness was a 34-percent decline in the occupancy of territories known to have falcons during the previous 10 years.

A high correlation was found also between the amount of DDE in eggs and eggshell thickness of pelican and double-crested cormorant eggs (Anderson et al., 1969).

DDE, a common breakdown product of DDT, fed to mallard ducks at 40 ppm (dry weight) induced a 14-percent (significant $P<0.01$) decrease in eggshell thickness (Heath, Spann and Kreitzer, 1969). Significant ($P<0.01$) eggshell thinning occurred even at 10 ppm of DDE. An important aspect of eggshell thinning was the eggshell cracking which resulted. DDE also caused a significant ($P<0.01$) reduction in percentage of "14-day ducklings of embryonated eggs" and number of "14-day ducklings per hen." Embryo mortality during the 4th week of incubation varying from 30 to 50 percent was attributed to DDE. Duckling production per hen was reduced as much as 75 percent when ducks were fed these levels of DDE.

DDT fed to ducks induced eggshell thinning at 25 ppm ($P=0.05$), but the effects were not as severe as those caused by DDE (Heath, Spann and Kreitzer, 1969). DDT fed at levels of 25 ppm reduced duckling survival approximately 35 percent ($P<0.01$).

Stickel and Rhodes (1969) reported that coturnix fed p,p'-DDT in their feed at dosages of 2.5 ppm, 10 ppm, and 25 ppm for 20 weeks produced overall 13 to 21 percent fewer eggs than did the control. Downward production trends continued for both the 10 ppm and 25 ppm dosages with time. Eggs produced by the DDT-treated birds had, respectively, 6.0, 6.4, and 7.3 percent thinner eggshells for 3 treatments than those of the untreated control birds. Although there was a tendency toward decreased hatchability of eggs from hens on the 25 ppm dosage, the decline was not statistically significant. However, "hatching success declined significantly ($P<0.025$) with time in all groups except those fed 2.5 ppm."

When mature coturnix were fed 0, 100, 200, and 400 ppm of DDT in their feed for 60 days, no effect on mortality, egg hatchability, or fertility was recorded for the 100 and 200 ppm dosages (Smith, Weber and Reid, 1969). The 400 ppm dosage within 30 days killed 50 percent of the quail and caused a marked decline in fertility and some decrease in hatchability. Young chicks hatching from eggs from the 400 ppm parents exhibited ataxia and spasms.

DDT and dieldrin fed in combination to American sparrow hawks under controlled conditions resulted in thinner eggshells (significance $P<0.01$) and increased egg disappearance (significance $P<0.05$) (Porter and Wiemeyer, 1969).

Ringdoves fed 10 ppm DDT showed a decrease of estradiol in the blood early in the breeding cycle, and egg-laying was delayed from a normal 16.5±1.6 days to 21.2±5.5 days (Peakall, 1970). The DDT caused about a 10-percent decrease in eggshell weight.

Bitman et al. (1969) reported that coturnix, fed high dosages of *o,p', and p,p'*-DDT, produced eggs with significantly less ($P<0.001$) calcium. Bitman, Cecil and Fries (1970) also reported that the shell-forming glands of coturnix fed DDT or DDE had carbonic anhydrase activity 16 to 19 percent lower than shell-glands of quail without pesticides. Both DDT and DDE fed to the quail caused about a 10-percent decrease in eggshell weight.

American sparrow hawks were fed for 2 years a diet containing 10 ppm p,p'-DDE on dry weight basis, a dosage equivalent to residue levels commonly found in the foods of raptors in the field (Wiemeyer and Porter, 1970). No difference in eggshell thickness was recorded between dosed and non-dosed birds during the first year, but "average shell thickness of eggs laid by DDE-dosed hawks in 1969 was 10 percent less than in 1968 (P<0.001)."

Field Studies. In Idaho bird populations in fir-pine forests treated with DDT at 1 lb/A appeared to be unaffected by the treatment (Adams et al., 1949). However, in Wyoming in similar type forests treated at 5 lb/A and 7½ lb/A the bird populations appeared to be suppressed, the greater suppression in the heavier dosage. Only a few dead birds were observed. Large numbers of invertebrate food organisms were killed in all the treated areas. Although there was no apparent immediate effect from this reduction, the long-term effects were not measured.

After the application of DDT at 2 lb/A every year for 4 years, populations of American redstarts, parula warblers, and red-eyed vireos in forested areas declined 44, 40, and 26 percent, respectively, over the 5-year experimental period, compared with the check area (Robbins, Springer and Webster, 1951). In the other 28 species of birds present in the areas little or no change in numbers was recorded.

The effect of 1 lb/A spraying of DDT for spruce budworm control on populations of 3 species of grouse (*Dendragapus obscurus, Bonasa umbellus, Canachites canadensis*) was investigated in Montana (Hoffman, Janson and Harthorn, 1958). Data on numbers of grouse, numbers of broods, and sizes of broods seen were available for the period from 1952 to 1956. No great change was noted in 1955, the year of spraying, or in the following year, in either sprayed or unsprayed areas. In another area sprayed with DDT in 1956, the proportion of young to adult grouse in hunter bags was as great as in unsprayed areas, suggesting no additional juvenile mortality resulting from this rate of DDT application.

Barker (1958) assayed soil and earthworms from beneath elm trees in a 450-acre area sprayed with 6-percent DDT for Dutch elm disease control and found that the soil contained up to 18 ppm DDT and/or DDE, and the earthworm species contained from 53 to 204 ppm (wet weight). Of the 21 dying robins collected in the treated area, the median residue in their bodies was 8 mg of DDT. Barker calculated that it would take fewer than 100 earthworms for a robin to accumulate the lethal dosage of 3 mg of DDT.

DDT applied to elms for control of Dutch elm disease resulted in a heavy mortality of robins and of many other species as well on the Michigan State University campus (Bernard, 1963, Bernard and Wallace, 1967; and Wallace, Etter and Osborne, 1964).

Three habitats in Wisconsin received DDT for control of Dutch elm disease, and 3 areas were unsprayed (Hunt, 1960). In the 3 DDT-treated habitats songbird numbers averaged 31, 68, and 90 percent below those of the unsprayed areas. Robin populations in the sprayed areas were 69, 70, and 98 percent below those of the unsprayed areas. Treatment of 2 areas in Wisconsin with DDT to control Dutch elm disease (about 2 pounds of DDT per tree) resulted in a robin mortality ranging from 86 to 88 percent (Hickey and Hunt, 1960). The number of nesting robins on the Madison, Wisconsin, campus increased from 3 pairs to 29 pairs after a change from DDT to methoxychlor (Hunt, 1965).

Elms were treated with DDT at 1.9 lb/A in Hanover, New Hampshire, resulting in 151 birds found dead, compared with untreated Norwich, Vermont, where only 10 birds were found dead (Wurster, Wurster and Strickland, 1965). The robin immigrant population in Hanover by June 1, 1963, had declined to 70 percent below the original May 1st population level. At Norwich there was no net change. Other birds affected included the myrtle warbler and the tree swallow.

The breeding success of New Brunswick woodcocks was closely related to the amount of DDT used (primarily for spruce-budworm control) in the summer range (Wright, 1965); an inverse relationship exists between breeding success and the amount of DDT used. From 1961 to 1963 the level of residues of DDT in spring woodcock arrivals in New Brunswick increased significantly from an average of 2.0 to 5.4 ppm DDT (Wright, 1965).

A DDT treatment of 1,768 acres of larch forest at a rate of 0.2 lb/A had no noticeable effect on the bird life (Schneider, 1966).

On the edge of Lake Michigan some mortality among herring gull adults was attributed to DDT present in the area (Keith, 1966a). Reproduction in these herring gulls appeared to be reduced by the presence of DDT. A sample of 9 eggs which appeared to be alive contained dosages of 202±84 wet weight ppm of DDE. The 10 dead eggs sampled had a higher concentration of 910±117 of DDE. From 30 to 35 percent of the eggs in 115 nests were dead, and this was felt to be an exceptional egg mortality.

In a rice-growing region in California where DDT-treated seed was used for pest control, pheasants were found to have concentrations of DDT averaging 740 ppm in their fat. The survival rate of young pheasants was lower than normal, prompting a restriction against planting DDT-treated seed (Hunt, 1966).

In an investigation of the effect of temperature and DDT spraying on the ruffed grouse population, Neave and Wright (1969) reported an apparent interaction between these 2 factors. May and June temperatures were related to the time of nest initiation, to egg loss, and to other mortalities. A synergistic effect between DDT (0.25 and 0.5 lb/A) and temperature was apparent in the loss of partially developed eggs. The DDT treatment was also correlated with a loss of immatures and changes in fall age ratios.

On Anacapa Island off the coast of California egg breakage resulted in the complete reproductive failure of the brown pelican on the island during 1969 (Keith, Woods and Hunt, 1970). Shells of a few intact eggs measured shortly after egg-laying averaged only 0.38 mm or were 34 percent thinner than normal (about 0.57 mm). Residues of DDT and its metabolites were about 1,200 ppm (85 percent DDE). Residues in the fat of adult birds ranged between 738 and 2,600 ppm. The authors concluded that "these findings, along with existing experimental evidence, clearly implicate DDE as a cause of eggshell thinning, reproductive failure, and population decline in brown pelicans."

On the east coast when eggshell weight and thickness of brown pelicans collected pre-1947 and in 1969 were compared, the eggshells had significantly ($P<0.01$) declined in both measurements (Blus, 1970). The author reported that the 16.9-percent decrease in eggshell weight of South Carolina pelican eggs approaches the level found in declining populations of several species of raptors.

Anderson and Hickey (1970) found that a small number (15) of pelican eggs taken in Texas and Florida after 1969 were 20 percent below normal weight. Also, 9 eggs from California collected in 1969 were 28 percent below normal weight. Shell thickness was found to have decreased between 15 and 37 percent.

Peakall (1970) reported that the "thin eggshell phenomenon" appears to be due to changes in "the storage and mobilization of calcium after ingestion, rather than action at the initial step of this process."

Fishes

Laboratory Experiments. The LC_{50} of DDT tested against various species of fish is found in table 11.

With increasing time and declining temperature the LC_{50} to DDT for rainbow trout decreased from 0.012 ppm to 0.0041 (table 12).

The relative toxicity of DDT to 3 species of fish, as measured by the 48-hour EC_{50}, was as follows: rainbow trout, 5 ppb (at 13°C); bluegill, 5 ppb (at 24°C); and channel catfish, 12 ppb (at 24°C) (Cope, 1966).

The 14-day LC_{50} for 10-week-old brown trout fry was 0.00086 ppm (King, 1962).

TABLE 11. The LC_{50} for various fish to DDT.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Brook trout	36	0.0023	Hatch, 1957
Landlocked salmon	36	0.08	"
Mosquito fish	96	0.09	"
Largemouth bass	96	0.002	Macek and McAllister, 1970
Brown trout	96	0.002	"
Coho salmon	96	0.004	"
Redear sunfish	96	0.005	"
Black bullhead	96	0.005	"
Rainbow trout	96	0.007	"
Bluegill	96	0.008	"
Yellow perch	96	0.009	"
Carp	96	0.010	"
Channel catfish	96	0.016	"
Fathead minnow	96	0.019	"
Goldfish	96	0.021	"
Goldfish	96	0.027	Henderson, Pickering and Tarzwell, 1959

TABLE 12. Effects of time and temperature on the toxicity of DDT to rainbow trout averaging approximately 1 g (Cope, 1965).

Temperature, °F	LC_{50} (ppb)		
	24 hrs	48 hrs	96 hrs
45	7.5	4.7	4.1
55	8.9	5.2	5.0
65	12.0	7.3	8.0

Guppies which had been exposed to sublethal doses of DDT for 14 days and then placed in a toxic concentration of DDT (0.032 ppm) demonstrated that they had increased their tolerance to the toxicant by this procedure (King, 1962).

Cutthroat trout were exposed in the laboratory for 30 minutes once a month for 1½ years to the following quantities of DDT in water baths: 0.01 ppm, 0.03 ppm, 0.1 ppm, 0.3 ppm, and 1.0 ppm (Allison et al., 1963). By the end of the experimental period from about 50 to 75 percent of the 650 fish in each group were dead at the 3 highest quantities of DDT. The number and volume of eggs produced by the trout were not reduced by these levels of DDT, but mortality among sac fry was higher at the 0.3 and 1.0 ppm DDT levels.

Mosquito fish collected from waters near cotton fields heavily treated with chlorinated insecticides exhibited significant levels of resistance to DDT, compared with fish from unexposed areas (Vinson, Boyd and Ferguson, 1963). A concentration of 0.05 ppm of DDT caused only about a 20-percent mortality in the resistant fish, whereas this same concentration caused about a 90-percent mortality in the susceptible fish.

In another study about 5 percent of mosquito fish surviving after exposure to DDT at concentrations above the threshold toxicity aborted their young (Boyd, 1964).

Ferguson et al. (1965a) collected resistant mosquito fish and black bullheads from streams in Mississippi and compared the toxicity in these fish to DDT with that in an unselected control population as measured by 36-hour LC_{50}. The results were: mosquito fish, control 80 ppb versus resistant (Sidon, Miss.) >200 ppb; and black bullhead, control 9 ppb versus resistant (Wayside, Miss.) 275 ppb.

Underyearling brook trout fed DDT at a rate of 2.0 mg/kg per week for 81 weeks gained more weight (40.9 g ±0.8 g) during the period than did the untreated controls (36.0 g ±1.1 g) (Macek, 1968a). When the underyearling trout were fed DDT at various rates for 26 weeks and then starved or fed at a rate equivalent to 10 percent of the usual feeding rate, the cumulative mortality during the experimental periods was 96.9 percent among fish fed DDT at 5.0 mg/kg per week, 86.6 percent for fish fed 2.0 mg/kg, and 1.9 percent for untreated fish. The author suggested that the mortality of DDT-exposed fish was due to the interaction of DDT with the starvation stress.

Brook trout behavior is also affected by sublethal doses (20 ppb) of DDT (Anderson and Peterson, 1969). Previously trained trout lost most of their learned avoidance response after their exposure to DDT. In addition, sublethal DDT doses (20 to 60 ppb) altered the thermal acclimation mechanism in brook trout.

Some species of fish are extremely sensitive to DDT. For example, the extrapolated LD_{50} dosage for young chinook and coho salmon was 0.0275 and 0.064 mg/kg/day, respectively. The chinook salmon appeared to be 2 to 3 times more sensitive to DDT than were coho salmon (Buhler, Rasmusson and Shanks, 1969).

Atlantic croakers were fed 2.57 μg of DDT per gram weight of fish for 67 days (Butler, 1966b). The accumulation of DDT resulted in mortality starting on the 14th day and continuing until all fish were dead by the 67th day.

Priester (1965) calculated the LC_{50} for the fathead minnow for 96-hour exposure to be 58 ppb of DDT. In young brook trout fed for 156 days with low concentrations of DDT, the major portion of the mortality (8 percent) occurred during the 15th week of development of the sac fry (Macek, 1968b).

DDT is not only toxic to fish but may also alter the normal behavior of fish. Ogilvie and Anderson (1965) found that New Bruns [illegible] salmon from a DDT-sprayed region were [illegible]ly sensitive to low temperatures and selected water of higher temperature than usual. If this response occurred in nature, salmon might place their eggs in regions where the young fry could not survive. *Gambusia* exposed to low levels of DDT (0.1 to 20 ppb) for 24 hours tended to prefer waters with a higher level of salinity than unexposed fish (Hansen, 1969).

The amount of DDT taken up by pinfish reached a maximum level about 2 weeks after exposure to dosages of 0.1 ppb and 1.0 ppb (Hansen, 1966).

At this time the pinfish had residues of about 8.8 ppm and 11.5 ppm.

DDT residues in coho salmon eggs from Lake Michigan measured during 1968 ranged from 1.1 to 2.8 ppm (Johnson and Pecor, 1969). Mortalities in the *fry* after hatching ranged from 15 to 73 percent. The higher residues of DDT in the eggs of these salmon were generally correlated with higher mortalities in the *fry*.

Field Studies. In Idaho and Wyoming treatment of forests with DDT at 1, 2½, 5, and 7½ lb/A influenced some fish populations (Adams et al., 1949). At the 1-lb/A dosage in Idaho, some sculpin (*Cottus beldingii*), mountain suckers, and black bullheads were killed by the DDT, but speckled dace, redside shiners (*Richardsonius balteatus hydrophlox*), rainbow trout, eastern brook trout, and cutthroat trout apparently were not affected. A few cutthroat trout were killed by the 2½ lb/A application of DDT. The most striking influence of DDT was on the diet of fish. Before treatment there were no crayfish in the diet, but immediately after the treatment the percentage increased to 99 percent, as in the case of the brook trout sampled. No measure of the long term effects of the change in food organisms was made in the investigation.

A spray calculated to give a DDT content of 0.09 ppm in water was used to treat a stream (Burden, 1956). Eight miles downstream from the treated area hundreds of fish were reported dying, and the concentration of DDT at a point 10 miles downstream was 0.017 ppm. "Two specimens of fish examined were found to have definitely died of poisoning * * *. These results lend support to previous work showing that fish are highly sensitive to DDT and that non-fatty animals are more sensitive than fatty ones" (Burden, 1956).

In 1955 when the fish hatchery on Lake George lost all of nearly 350,000 eggs removed from lake trout, DDT was suspected as the cause. For several years about 10,000 pounds of DDT had been distributed yearly for control of gypsy moth and biting flies in the watershed associated with Lake George (Burdick et al., 1964).

Careful studies revealed that DDT stopped reproduction of lake trout in Lake George and several other heavily contaminated lakes in the adjacent Adirondack region. Although the trout eggs contained from 3 to 355 ppm of DDT, little or no mortality occurred in the egg stage. The fry, however, were highly sensitive to these dosages and were killed at the time of final absorption of the yolk sac, just when they were ready to feed. For example, at levels of DDT in eggs that would produce 3 ppm in fry, few fry survived, and at 5 ppm DDT none survived (Burdick et al., 1964).

The spraying of New Brunswick forests with DDT between 1958 and 1960 was reported by Elson and Kerswill (1964) to be responsible for the severe reduction in salmon fishing success in the province, especially between 1959 and 1962.

DDT was applied at 1 lb/A to about 72,000 acres in the Yellowstone River drainage in 1957 for spruce budworm control, and the effects on various non-target organisms were recorded (Cope, 1961). DDT was found up to 0.08 ppm in the water, and in one case a trace was found 55 miles downstream from the treated area. Samples of mountain whitefish, rainbow trout, and brown trout contained DDT up to 14.00 ppm or DDE up to 6.55 ppm or both. The author further reported that "DDT was found in trout 88 miles below the spray area, and fish taken more than 2 years after spraying contained DDT."

DDT was applied at 0.2 lb/A to a tidal marsh in Florida (Croker and Wilson, 1965). Total kills of caged striped mullet, sheepshead, longnose killifish, rainwater killifish, and tidewater silverside occurred in 1 to 24 days. Fish accumulated up to 90 ppm of DDT within 5 weeks after treatment.

Applications of DDT to control nuisance insects appeared to be associated with the decline of the salmon fishery at Sebago Lake, Maine (Anderson and Everhart, 1966). Average DDT residues in salmon collected in 1962, 1963, and 1964 (10 each year) were 1.1, 3.2, and 1.8 ppm by total weight. Salmon in the 3-year age group had 1.2 ppm, 4-year age group had 8.0 ppm, and 5-year age group had 8.8 ppm of DDT.

Cuerrier, Keith and Stone (1967) reported that when levels of DDT and its metabolites were above 400 ppb in the eggs of hatchery trout, the "mortality in the resulting fry ranged from 30 percent to 90 percent in the 60-day period following the swim-up stage."

The mortality of young Atlantic salmon and eastern brook trout was observed in cages and free-living in streams in forested areas of New Brunswick sprayed with DDT for spruce budworm control (Kerswill and Edwards, 1967). There were no short-term effects on salmon parr with DDT at ½ lb/A, but many yearlings were killed. Two applications of ¼ lb/A 10 days apart were as harmful as a single application of ½ lb/A. DDT at ½ lb/A caused a heavy loss (50 to 98 percent) of underyearling and parr salmon (Elson, 1967).

The application of DDT at ½ lb/A to a forest watershed of the Northwest Miramichi River, New Brunswick, changed the kinds of food found in stomachs of young Atlantic salmon (Keenleyside, 1967). Salmon under one year typically consume immature aquatic Diptera and small Ephemeroptera insects, whereas salmon over one year consume Diptera, Trichoptera, and all sizes of Ephemeroptera. After the DDT application the surviving young salmon fed on the resurgence of Chironomidae and other Diptera; the salmon more than one year fed on Diptera, worms, snails, and fish which previously had been unimportant in their diet. Salmon feeding on aquatic organisms approached pre-spray normal species complexity 5 years after the last application.

Observations in the field confirm laboratory findings that DDT is highly toxic to some fish and especially to fry. DDT residues in coho salmon eggs from Lake Michigan measured during 1968 ranged from 1.1 to 2.8 ppm. Mortalities in the fry after hatching ranged from 15 to 73 percent. The higher residues of DDT in the eggs of these salmon were generally correlated with higher mortalities in the fry (Johnson and Pecor, 1969); however, it should be pointed out that dieldrin residues were also detected in the fish.

Amphibians and Reptiles

The LD_{50} for bullfrogs was >2,000 mg/kg to DDT when the frogs were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

In 1951 one pound of DDT per acre was applied for control of tent caterpillars in Hubbard County, Minnesota (Fashingbauer, 1957). Before spraying 111 small *Rana sylvatica* were counted around 2 pools. The frogs seemed well a day after spraying, but the water had oil film and was covered with poisoned caterpillars. Two and a half days later 35 dead frogs were found, and after a few more days no living ones remained. All but 2 of 34 frog stomachs contained tent caterpillars, among other insects. Whether frogs were killed directly or indirectly by eating poisoned insects, the local population was drastically reduced.

Boyd, Vinson and Ferguson (1963) demonstrated the presence of resistance to DDT in natural populations of cricket frogs (*Acris crepitans* and *A. gryllus*) from heavily treated cotton-field areas. The mortality data for the 2 species from untreated areas were generally higher than in frog populations having a history of exposure to DDT.

The 24-hour LC_{50} for Fowler's toad tadpoles and chorus frog tadpoles exposed to DDT was 2.4 ppm and 1.4 ppm, respectively (Sanders, 1970).

Box turtle populations were not noticeably affected by DDT applied at a rate of 2 lb/A to a forest in Maryland (Stickel, 1951).

Molluscs

DDT content in seawater at 0.1 ppm halted the growth of eastern oysters, and dosages as low as 0.0001 ppm significantly reduced oyster growth (Butler, 1966b).

Eastern oysters containing about 151 ppm of DDT required approximately 3 months in clean water to lose 95 percent of their load of DDT. Their growth returned to normal after only 10 days of flushing in clean water (Butler, 1966a). Several other mollusc species lost about 75 percent of accumulated DDT after 15 days of flushing in clean water (table 13).

Arthropods and Annelids

The toxicity of DDT to insects and crustaceans as measured by a 48-hour EC_{50} was as follows: stonefly nymph (*Pteronarcys californicus* [*sic*]) at 10 ppb, mayfly nymph (*Baetis* sp.) at 19 ppb, waterflea (*Simocephalus serrulatus*) at 0.4 ppb, and waterflea (*Daphnia pulex*) at 9 ppb (Cope, 1966).

The LC_{50} of DDT tested against various species of arthropods is found in table 14.

Ine 4 predators and parasites (syrphids, coccinellids, and braconids) were more susceptible to DDT than the cabbage aphid (Way, 1949). The parasitic braconids were especially sensitive.

Hueck et al. (1952) observed an increase in egg production in fruit-tree-red spiders (*Metatetranychus ulmi*) after exposure to low concentrations of DDT, but later research by Pielou (1960) did not substantiate this finding.

TABLE 13. Accumulation and retention of DDT by molluscs exposed for 7 days to 1.0 μg/l in flowing seawater and then placed in clean water (Butler, 1966a).

Mollusc	Residue (ppm)		
	After 7 Days Exposure	After 15 Days Exposure	After 30 Days Exposure
Hooked mussel	24		
Eastern oyster	26	2.5	1.0
Pacific oyster	20	16.0	
European oyster	15	8.0	4.0
Crested oyster	27	5.0	
Northern quahog	6	0.5	

Populations of the mite (*Tetranychus bimaculatus*) on beans and potatoes increased up to 20 times the density of the untreated control after applications of DDT at dosages from 10 to 119 lb/A (Klostermeyer and Rasmussen, 1953).

Outbreaks of two-spotted mites (*Tetranychus telarius*) were observed in peach orchards after the use of DDT. DDT was suggested to have killed predators of the mites (Pickett, Putman and Leroux, 1958).

In orchards DDT applied for the control of apple pests eliminated populations of certain highly susceptible, predaceous ladybird beetles (Hello, 1955). As these beetles were the principal controlling agent for a red-mite pest, the mite population subsequently reached outbreak levels, causing severe damage to the apple trees. This particular mite is not susceptible to DDT and was therefore hardly influenced by the chemical which killed the beetle.

DDT suppressed the ovarian development in the housefly (Beard, 1965). Weevils (*Sitophilus granarius*) exposed to 0.10 and 0.125 mg of DDT per 100 g wheat produced 90 percent more offspring than unexposed weevils (Kuenen, 1958).

Outbreaks of the red-banded leaf roller occurred in apple orchards after the use of DDT because the leaf roller's parasites and predators were more susceptible than the leaf roller (Paradis, 1956).

DDT treatment of cole crops resulted in aphid outbreaks, probably due to predator and parasite destruction (Pimentel, 1961). Although the number of predators and parasites was larger in the DDT-treated area than in the untreated control (presumably because they were attracted by the aphid outbreak), the ratio of predators and parasites to aphids was nevertheless significantly lower in the DDT-treated area.

The evidence suggests that DDT is highly toxic to some invertebrates, whereas others are relatively resistant. Priester (1965) recorded the 48-hour LC_{50} for *Daphnia* as 1.48 ppb of DDT, indicating that they are quite sensitive. DDT at concentrations of 1 to 6 ppb also killed or immobilized 50 percent of the brown and pink shrimp exposed for 48 hours in laboratory tests (Butler and Springer, 1963). The LC_{50} to DDT for red crawfish at 48 hours was 0.5 ppm (Muncy and Oliver, 1963).

DDT caused a reduction in numbers of natural predators, followed by an increase in numbers of European red mites and clover mites. The concentration of DDT used and the time applied determined the frequency and magnitude of population outbreaks attained by the mites, the reestablishment of predator populations, and time required for reattainment of equilibrium of low populations of predators and mites (Lord, 1956).

TABLE 14. The LC_{50} for various arthropods to DDT.

Formulation	Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
P.D.[1]	Sand shrimp	96	0.0006	Eisler, 1969
	Amphipod (*Gammarus lacustris*)	96	0.0047	Sanders, 1969
P.D.[1]	Hermit crab	96	0.007	Eisler, 1969
P.D.[1]	Grass shrimp	96	0.010	"
	Stonefly (*Pteronarcella badia*)	96	0.019	Sanders and Cope, 1968
	" (*Claassenia sabulosa*)	96	0.016	"
	" (*Pteronarcys californica*)	96	0.061	"
	Waterflea (*Daphnia pulex*)	48	0.00036	Sanders and Cope, 1966
	" (*D. pulex*)	48	0.0036	FWPCA, 1968
	Amphipod (*G. lacustris*)	48	0.0031	"
	Stonefly (*P. californica*)	48	0.019	Sanders and Cope, 1968
	" (*P. californica*)	48	0.019	FWPCA, 1968

After DDT treatments for codling moth control, the European red mite was abundant, whereas its predator (*Stethorus punctum*) was absent (Steiner, Arnold and Summerland, 1944). An increase of the Pacific spider mite on trees treated with DDT was attributed to the destruction of natural enemies (Newcomer and Dean, 1946).

In apple orchards, also, DDT applications sharply reduced the parasitism of the apple mealy bug, as DDT is highly toxic to its parasite (*Pseudaphycus*) (Hough, Clancy and Pollard, 1945).

The oriental fruit moth showed a marked decrease of parasitism by the braconid (*Macrocentrus ancylivora*) with the use of DDT in peach orchards in New York (Wheeler and LaPlante, 1946). In untreated orchards the parasitism rate of the moth was 51.8 percent, compared with only 10.8 percent in treated orchards.

When DDT was widely used on citrus and grapes and for mosquito control, it caused an outbreak in the cottony-cushion scale because the vedalia coccinellid predator was destroyed (DeBach, 1947).

In California applications of DDT favored the increase of yellow scale by killing its encyrtid parasite, *Comperiella bifasciata*. On the other hand, applications of DDT dust did not observably affect *Metaphycus luteolus*, a parasite of the citricola scale (Woglum et al., 1947).

Ide (1967) investigated the number of aquatic insects present in the forest-covered tributaries of the Miramichi River of northern New Brunswick after aerial treatment with 0.5 lb/A of DDT. In the streams affected by DDT fewer insect species emerged, and those species most severely reduced were the larger ones, such as caddice flies. The treated streams generally had larger numbers of individuals, but the weight of insect life was in some cases reduced by half. Furthermore, the insect fauna of the treated streams were deficient in the species of insects on which salmon mainly feed. From 2 to 3 years were necessary for the fauna to recover qualitatively for most groups; however, for some recovery required 4 years (Ide, 1967).

One spraying of forests at the rate of 5 lb/A of DDT destroyed many natural enemies, resulting in outbreaks of both aphids and mites (Hoffman and Merkel, 1948). These outbreaks, however, were of relatively short duration.

Vo'hers and Carlson (1948) observed predaceous coccinellid insects to be repelled by DDT. Some coccinellids which did enter DDT-treated plots were destroyed, and those surviving had reduced oviposition rates.

Coccinellid beetles were also found capable of resisting DDT, in part because of their ability to metabolize DDT to DDE and to excrete these 2 compounds in their feces and eggs (Atallah and Nettles, 1966).

After the application of DDT at 0.25 lb/A to a small stream in Pennsylvania, about 90 percent of the total stream insect population was apparently exterminated, and about one-third of the species eliminated (Hoffmann et al., 1946). Some species did not repopulate the stream for 2 years or more.

DDT was applied at 1.0 lb/A for control of the spruce budworm to the Swan Creek drainage area in Montana (Bridges and Andrews, 1961). Although the spraying aircraft did not treat within ¼ of a mile of the stream, 0.01 ppm of DDT was measured in the water ½ hour after spraying. Three hours after treatment samples of insects contained up to 11 ppm. Extreme mortalities occurred in mayfly nymphs, caddice fly larvae, and stonefly larvae by one hour after treatment. Rainbow trout in the creek suffered no acute effects.

In Georgia, DDT was applied at 0.5 lb/A for control of the elm spanworm (Frey, 1961). In the one drainage area where precautions were not taken to avoid the stream, a serious kill of mayfly and stonefly nymphs occurred (about a 90-percent reduction). Recovery of the bottom invertebrates was rapid, and within 4 months after treatment invertebrate numbers were back to pre-treatment levels.

After the treatment of 72,000 acres of the Yellowstone River system with DDT at 1 lb/A (as mentioned), stream-bottom invertebrates were significantly reduced in number (Cope, 1961). Total numbers of invertebrates had recovered within a year, but the species composition was still altered. Both the Plecoptera and Ephemeroptera were reduced, but both Trichoptera and Diptera occurred at higher numbers at the end of one year.

When DDT was employed for malaria control in Sardinia, *Anopheles labranchiae* was effectively controlled, but then 2 rare species (*A. claviger* and *A. algeriensis*) increased to replace the eradicated *A. labranchiae* (Aitken and Trapido, 1961). Such replacement may or may not be detrimental.

Because of environmental exposure to DDT and the resulting selective pressures, honeybees at

Riverside, California, were found to be 6 times more resistant to DDT than honeybees from unexposed areas (Atkins and Anderson, 1962). These results provide an idea of the amount of DDT in the environment and intensity of selective pressure.

After the application of DDT for the control of caterpillars, in particular *Pieris rapae*, on cole crops, Dempster (1968b) reported that the survival of the pest was better than expected because the insecticide killed many of the caterpillars' natural enemies. Dempster (1968c) indicated that it was impossible to predict the changes in species populations with the application of any one insecticide to a biotic community. However, one common trend was the reduction or elimination of natural enemies, frequently leading to outbreaks in the numbers of herbivores or pest species on the cole crop under study.

DDT applied at 25 lb/A reduced earthworm activity, as measured by castings, by 80 percent (Doane, 1962). DDT at 37.2 lb/A reportedly caused significant reductions (43 percent) in the number of earthworms in golf courses in Ohio (Polivka, 1951).

Earthworm populations were found to reflect the dosage of DDT in soil (Stringer and Pickard, 1964). In soils containing 26.6 ppm, 4.1 ppm, and 8.6 ppm the earthworms (*Lumbricus terrestris* and other species) in these soils averaged about 4 ppm, 7 ppm, and 5 ppm, respectively.

Dempster (1968a) found that low concentrations of DDT significantly reduced the rate of adult feeding in a predaceous ground beetle.

Menhinick (1962) compared invertebrate populations in orchard litter and soil contaminated with DDT and other pesticides to those in areas free of pesticides and found the diversity of species and total biomass of living organisms lower in the contaminated area, but the numbers of individuals much higher. For example, tremendous numbers of Collembola, sarcoptiform mites, and aphids were present in contaminated areas; however, the larger invertebrate predators like beetles and flies were significantly reduced in numbers in the contaminated area.

DDT applied at 200 lb/A of 5-percent dust to year-old fallow plots (Edwards, Dennis and Empson, 1967) did not affect the Lumbricidae, Enchytraeidae, or Nematoda, but did significantly reduce the monostigmatid mites, apparently causing an increase in most Collembola species populations. Both Coleoptera and Diptera biomass were reduced. In general, the DDT killed more pest species than predaceous or beneficial species.

DDT larviciding at 0.1 ppm for control of black flies in Bobby's Brook, Labrador, resulted in several faunal changes (Hatfield, 1969). Caddice fly larval populations were reduced to zero or near zero at all stations receiving the treatment, and the same was true for stonefly and mayfly larvae. The DDT also caused mortalities in eastern brook trout by contamination of the fish foods above maximum tolerance levels. The treatment, however, caused no significant short-term fish mortality by direct contact.

In greenhouse tests alfalfa treated with DDT at 1 lb/A after 11 days' exposure caused about a 70-percent mortality in pollinating leafcutting bees (Waller, 1969).

Brown (1969) reported that 225 species of insects and mites have evolved resistance to DDT, Cyclodiene, and organophosphorus insecticides. The 225 species were broken down as follows: 121 crop pests, 97 man and animal pests, 6 stored-product pests, and 1 forest pest.

Fiddler crabs fed natural organic plant detritus for 11 days in estuaries containing DDT (10 ppm) exhibited grossly modified behavior (Odum, Woodwell and Wurster, 1969). Within 5 days on the DDT containing detritus the crabs became uncoordinated. When threatened, they did not scurry way, but moved a short distance, lost coordination and equilibrium and rolled over.

Plants

Dosages of DDT applied at 24 lb/A and above significantly depressed the growth of rye and proved highly toxic to beans (Boswell et al., 1955).

The effect of DDT added to the soil of orchards annually at 200 lb/A from 1949 to 1953 was measured by growing various crop plants in the contaminated soil for several years following the treatments (MacPhee, Chisholm and MacEachern, 1960). With a residue in the soil of about 110 ppm of DDT at time of growth, yields of the crop plants were as follows: beans, reduced by 66 percent; turnips, no effect; carrots, reduced by 40 percent; tomatoes, reduced by 93 percent; and peas, reduced by 33 percent.

Only rates of 24 lb/A and higher of DDT reduced the growth of stringless black valentine beans during the same year as application (Clore et al., 1961).

DDT at a 0.5-percent aqueous solution killed most rye plants, but a small percentage (2.58) were found to be resistant (Jones and Hayes, 1967). These surviving plants had a significant level of resistance to DDT.

Corn grown in DDT-treated soil at 10 and 100 ppm weighed significantly more (9 g at 100 ppm) than untreated corn (6.5 g) at the end of 4 weeks; however, after 8 weeks of growth this difference was no longer detectable (Cole et al., 1968). Beans, on the other hand, weighed significantly less when exposed to DDT concentrations of 1 ppm (8.3 g) and 10 ppm (8.7 g) for 8 weeks than the untreated (9.7 g).

DDT at 1, 10, and 100 ppm in the soil caused significant changes in the macro and micro element (N, P, K, Ca, Mg, Fe, Cu, B, Al, Sr, and Zn) constituents of above-ground portions of corn and bean plants (Cole et al., 1968). For example, the manganese content in beans was significantly higher in the DDT (100 ppm)-treated, 512 ppm dry weight, than the control, 380 ppm, at the end of 8 weeks' growth; however, the boron content was lower in the DDT (100 ppm)-treated, 17 ppm, than the control, 28 ppm, at the end of 8 weeks' growth.

Exposing phytoplankton communities in the laboratory for 4 hours to 1 ppm of DDT in water reduced their productivity 77.2 percent (Butler, 1963a). DDT at about 0.01 ppm reduced photosynthesis in laboratory cultures of 4 species of coastal and oceanic phytoplankton (*Skeletonema costatum, Coccolithus huxleyi, Pyramimonas* sp., *Peridium trochoideum*) and a natural phytoplankton culture from Woods Hole, Massachusetts (Wurster, 1968). These exposure levels are far higher than those likely to be achieved in the sea. Christie (1969), however, reported that freshwater algae was not affected at a high dosage of DDT (100 ppm).

Södergren (1968) reported that the uptake of DDT by phytoplankton (*Chlorella* sp.) was extremely rapid; the process was completed in less than 15 seconds. Significant morphological and physiological changes were noted in the phytoplankton after growing in presence of DDT at less than 0.5 ppb for 3 days.

Biological Concentration

Eastern oysters placed in flowing seawater containing 0.1 ppb of DDT for 40 days concentrated DDT some 70,000 times the level in the water (Butler, 1964). Oysters exposed for 10 days to a mixture of 8 pesticides in the water, ranging from 0.001 to 0.05 ppm, increased the pesticide concentrations in their bodies; DDT, for example, was concentrated 15,000 times (Wilson, 1965).

A saltwater fish (croakers) concentrated DDT 20,000 times the level in water (0.001 ppm) (Hansen, 1966). After 2 weeks of exposure to 0.001 ppm of DDT in water, 10 fish concentrated the level of DDT in their bodies 12,000 times the level of the water. When 10 fish were exposed to a lower concentration of DDT (0.0001 ppm), they were found to be able to concentrate the level in their own bodies 40,000 times that of the water.

DDT residues were found to reach a level of more than 13 lb/A in Long Island saltmarsh (Woodwell, Wurster and Isaacson, 1967). In a sampling of the marsh and organisms present in the saltmarsh DDT in the water was estimated at 0.05 ppb and in plankton the level was 40 ppb of DDT. The highest concentrations were detected in the scavenging and carnivorous fish and birds; the birds were reported to have 10 to 100 times more than the fish species.

Samples removed from a tidal marsh habitat in Florida treated with 0.2 lb/A of DDT contained the following levels of DDT: surface water and ditch, 0.5 to 4.04 ppm; sediment samples, as high as 3.5 ppm (dry weight); vegetation, as high as 75 ppm (dry weight); and in 5 species of fish DDT ranged from 2 to 58 ppm (wet weight) (Croker and Wilson, 1965).

In Lake Michigan sediments on a wet weight basis averaged 0.014 ppm of DDT, DDE, and TDE. From the same habitat the amphipod *Pontoporeia affinis* averaged 0.41 ppm for DDT and its related metabolites, or about 30 times the level found in the mud; various fish removed from the lake had residues of 3.35 ppm (alewives), 4.52 ppm (chub), and 5.60 ppm (whitefish), or about 10 times that of the amphipod; and in the gulls, breast muscle averaged 27 times the level of DDT found in the alewives (Hickey, Keith and Coon, 1966). Body fat of the gulls averaged 2,441 ppm DDT.

In ponds containing 0.02 ppm of DDT in water, rainbow trout, black bullhead, and crayfish were found to concentrate DDT to the levels of 4.15 ppm, 5.11 ppm, and 1.47 ppm, respectively (Cope, 1966).

The chemical attributes of DDT make it susceptible to biological concentration in algal living systems. For example, 4 species of algae concentrated DDT about 220-fold when exposed to a concentration of DDT at 1 ppm in water for 7 days (Vance and Drummond, 1969). *Daphnia*, a zooplanktonic organism, concentrated DDT 100,000-fold during a 14-day exposure to water containing 0.5 ppb of DDT (Priester, 1965). A fathead minnow concentrated DDT further in its tissues on being fed *Daphnia* containing DDT (Priester, 1965).

In some DDT-sprayed elm environments pesticide residues accumulated from 9.9 ppm in the soil to 141 ppm in earthworms to 444 in the brain of adult robins (Hunt, 1965). In another area where elm trees had been sprayed with DDT for control of Dutch elm disease, the soils had a residue to 10 ppm of DDT and earthworms from the same soil contained 157 ppm (Hunt, 1965).

Diamond et al. (1970) demonstrated that forest soil with a mean DDT residue of about 1 ppm resulted in the earthworms having residues between 0.10 and 0.52 ppm (wet weight). Robins in this forest had DDT residues ranging from 2.26 to 15.38 ppm (wet weight).

Slugs and earthworms in a cotton field concentrated DDT 18 and 11 times the level in the soil, respectively; the slugs contained 58 ppm of DDT and its metabolites, and the earthworms contained 59 ppm (USDI, 1965).

Kinetics

The kinetics of DDT loss from organisms has been reported for a few species. For example, when oysters which had accumulated a body burden of 150 ppm of DDT were placed in clean water, they lost two-thirds of this concentration in 60 days, but required 40 more days before the residue levels decreased to 5 ppm (Butler, 1966b).

An investigation of DDT residues in fish in the Farmington and Connecticut River Watersheds indicated that the DDT residues in the fish declined significantly from the fall of 1960, when DDT applications were halted, to the spring of 1964 (Tompkins, 1964).

Of interest was the report that migratory birds lose 50 percent of their body burden of DDT during their movement from Mexico to Canada (Harvey, 1967).

One means by which DDT may get into the air from water is codistillation of DDT with the water (Acree, Beroza and Bowman, 1963). They also suggested that DDT appears to concentrate at the water surface, thus providing a better opportunity for DDT to escape the water medium and enter the air. The full significance of this finding has not been investigated.

Fish reach some equilibrium between the amount of insecticide in the environment and the amount in the organism (Hansen, 1966 in Dustman and Stickel, 1969). Pinfish, for example, when exposed to 0.001 ppm of DDT in the water environment reached an equilibrium within 9 weeks, at which time body residues were 12 ppm, or 12,000 times that of the environment. In another experiment at a lower dosage in the water, 0.0001 ppm, the equilibrium was achieved in the same time but the dosage in the fish was 4 ppm, or 40,000 times that of the environment.

Persistence

DDT applied at 10 to 20 lb/A persisted in soil for >4 years (Allen et al., 1954) to ≥10 years (Clore et al., 1961).

The percentage of DDT applied at a rate of 100 ppm to sandy loam soil remaining after 17 years was 39 percent (Nash and Woolson, 1967).

Soil residues in a Maine forest treated with DDT at 1 lb/A showed little decrease during the 9 years after application (Diamond et al., 1970). The investigators suggested that the residues may persist for 30 years.

DELMETON

Fishes

The 48-hour LC_{50} for bluegill exposed to delmeton was 81 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for waterfleas (*Daphnia pulex*) exposed to demeton was 14 ppb (FWPCA, 1968).

DEMETON

Mammals

The LD_{50} for the rat was 1.7 (demeton S) and 7.5 (demeton O) (USDI, 1970a) and for domestic goats, 8 to 16 mg/kg (Tucker and Crabtree, 1970) to demeton when the mammals were given the stated dosages orally in a capsule.

Birds

The LD_{50} for young mallards was 7.2 mg/kg; for young pheasants, 8.9 mg/kg; for young chukar partridges, 15.1 mg/kg; for young coturnix, 6.5 mg/kg; for pigeons (*Columba livia*), 8.5 mg/kg; for sharp-tailed grouse, 4.8 mg/kg; for house sparrows, 9.5 mg/kg; and for house finches, 2.4 mg/kg to demeton when the birds were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970). The LC_{50} for mallards was 600 to 700 ppm; for pheasants, 650 to 700 ppm; for bobwhites, 550 to 650 ppm; and for coturnix, 200 to 300 ppm of demeton in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

Amphibians

The LD_{50} for bullfrogs was 562 mg/kg to demeton when the frogs were fed the stated dosage orally in capsules (Tucker and Crabtree, 1970).

Persistence

Demeton in soil persisted for 28 days (Laygo and Schulz, 1963).

DEMETON METHYL

Birds

Demeton methyl in acetone injected into hen eggs at 10 ppm, 50 ppm, and 100 ppm killed 5, 16, and 70 percent of the embryos (Dunachie and Fletcher, 1969). This toxicant also caused teratogenic effects at 100 ppm.

Fishes

The 24-hour LC_{50} for harlequin fish to demeton methyl was 9 ppm (Alabaster, 1969).

DIAZINON

Mammals

The LD_{50} for the rat was 76 to 108 mg/kg to diazinon when the mammals were fed the stated dosages orally (Neumeyer, Gibbons and Trask, 1969).

Birds

The LD_{50} for young mallards was 3.5 mg/kg and for young pheasants, 4.3 mg/kg to diazinon when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Diazinon applied at the rate of 5 lb/A killed nearly 50 percent of the pheasant population in the test field area (USDI, 1967). No deaths were recorded at the 1-lb/A level.

Fishes and Mussels

The LC_{50} for bluegills and rainbow trout to diazinon for a 24-hour exposure was 0.052 ppm (75°F) and 0.380 ppm (55°F), respectively (Cope, 1965).

The 48-hour LC_{50} for bluegill exposed to diazinon was 30 ppb (FWPCA, 1968).

The relative toxicity of diazinon to 9 species of fish as measured by a 48-hour EC_{50} was as follows:

rainbow trout at 170 ppb, 13°C; and bluegills at 60 ppb, 24°C (Cope, 1966).

The 24-hour LC_{50} for harlequin fish to diazinon was 1.45 ppm (Alabaster, 1969).

Diazinon applied at a rate of 0.32 ppm to a cranberry bog disappeared completely from the water within 144 hours. The water immediately after treatment was toxic to fish (*Fundulus heteroclitus*), which concentrated the diazinon to a level about 10 times that of the surrounding water. Freshwater mussel (*Elliptio complanatus*) survived the exposure to diazinon, yet concentrated the material by about twice the levels in the surrounding water (Miller, Zuckerman and Charig, 1966).

An investigation of the persistence of diazinon in fish revealed that 50 percent of the chemical was lost in less than 1 week (Miller, Zuckerman and Charig, 1966).

Amphibians

The LD_{50} for bullfrogs was >2,000 mg/kg to diazinon when the frogs were fed the stated dosage orally (Tucker and Crabtree, 1970).

Arthropods

The LC_{50} for various arthropods to diazinon is found in table 15.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to diazinon was 1.8 ppb and 0.90 ppb, respectively (Sanders and Cope, 1966).

Detectable levels of diazinon were found in the soil up to 84 days after treatment with 14 lb/A. The insect populations decreased after the initial treatment, but then there was a rapid resurgence of many of the species. Interestingly enough, there were also significant changes in the vegetation involving the abandoned field; both species diversity and density increased in the treated area (Malone, Winnett and Helrich, 1967).

Biological Concentration

Fish (*F. heteroclitus*) concentrated diazinon to a level about 10 times that in the surrounding water (0.32 ppm) (Miller, Zuckerman and Charig, 1966).

Persistence

Diazinon applied to soil persisted at detectable levels for 9 days (Laygo and Schulz, 1963) and for about 12 weeks (Kearney, Nash and Isensee, 1969).

DIBROMOCHLOROPROPANE

Mammals

The LD_{50} for the rat was 173 mg/kg and for the mouse, 257 mg/kg to dibromochloropropane when the mammals were fed the stated dosages orally (FCH, 1970).

Birds

The LD_{50} for young mallards was 66.5 mg/kg to dibromochloropropane when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

TABLE 15. The LC_{50} for various arthropods to diazinon

Arthropod Species	Exposure Hours	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	96	0.200	Sanders, 1969
Waterflea (*Daphnia pulex*)	48	0.0009	Cope, 1966
" (*D. pulex*)	48	0.0009	FWPCA, 1968
Stonefly (*Pteronarcys californica*)	48	0.060	"
" (*P. californicus* [sic])	48	0.074	Cope, 1966
Amphipod (*G. lacustris*)	48	0.500	FWPCA, 1968

Mollusca

Davis (1961) reported that dibromochloropropane at concentrations of 1 ppm and above caused a 90-percent mortality in clam larvae after 24 hours of exposure.

Annelids

No earthworms survived 1 day in pots containing soil treated with 20 gal/A of dibromochloropropane in the laboratory (DeVries, 1962). After 33 days 87 percent of the *Lumbricus* and 28 percent of the *Helodrilus* were killed with a dosage of 8 lb/A.

DICAPTHON

Mammals

The LD_{50} for rats was 400 mg/kg to dicapthon when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

When chickens were fed dicapthon at a dosage of 200 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

DICHLOFENTHION

Mammals

The LD_{50} for the rat was 250 mg/kg to dichlofenthion when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to dichlorphenthion (summer sheep dip) was 3.2 ppm (Alabaster, 1969).

DICHLORVOS

Mammals

The LD_{50} for rats was 56 to 80 mg/kg (Neumeyer, Gibbons and Trask, 1969) and for mice, 4 mg/kg (Negherbon, 1959).

Birds

The LD_{50} for young mallards was 7.8 mg/kg and for young pheasants, 11.3 mg/kg to dichlorvos when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was >5,000 ppm to dichlorvos in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Dichlorvos in acetone injected into hen eggs at 50 ppm, 100 ppm, and 200 ppm killed 22, 45, and 40 percent of the embryos (Dunachie and Fletcher, 1969).

Fishes

The LC_{50} for bluegills to dichlorvos for 24-hour exposure was 1 ppm (Cope, 1965).

The 48-hour LC_{50} for bluegill exposed to dichlorvos was 700 ppb (FWPCA, 1968).

The 24-hour LC_{50} for harlequin fish to dichlorvos was 10 ppm (Alabaster, 1969).

Arthropods

The LC_{50} for various arthropods to dichlorvos is found in table 16.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to dichlorvos was 0.26 ppb and 0.066 ppb, respectively (Sanders and Cope, 1966).

Persistence

The persistence of dichlorvos at detectable levels in water at 20°C was 62 days (Muhlmann and Schrader, 1957).

TABLE 18. The LC_{50} for various arthropods to dichlorvos.

Arthropod Species	Exposure Period (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	0.004	Sanders, 1969
Sand shrimp	24	0.015	Eisler, 1969
Stonefly (*Pteronarcys* sp.)	24	0.003	Cope, 1965
" (*P. californica*)	24	0.035	Sanders and Cope, 1968
Hermit crab	24	0.150	Eisler, 1969
Grass shrimp	24	0.180	"
Waterflea (*Daphnia pulex*)	48	0.00007	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.001	"
Stonefly (*P. californica*)	48	0.010	"

DICOFOL

Mammals

The LD_{50} for the rat was 700 mg/kg to dicofol when the mammals were fed the stated dosage orally (Metcalf, Flint and Metcalf, 1962).

Birds

The LC_{50} for mallards was 1,700 to 1,900 ppm; for pheasants, 2,100 to 2,300 ppm; for bobwhites, 2,800 to 3,300 ppm; and for coturnix, 1,400 to 1,600 ppm of dicofol in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Dicofol in acetone injected into hen eggs at 500 ppm killed only 30 percent of the embryos (Dunachie and Fletcher, 1969).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to dicofol was 100 ppm (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for stoneflies (*Pteronarcys californica*) and waterfleas (*Daphnia magna*) exposed to dicofol was 3,000 ppm and 390 ppm, respectively (FWPCA, 1968).

DICROTOPHOS

Mammals

The LD_{50} for rats was approximately 22 mg/kg and for mice, 15 mg/kg to dicrotophos when the mammals were fed the stated dosages orally (FCH, 1970).

Birds

The LD_{50} for young mallards was 4.2 mg/kg; for young pheasants, 3.2 mg/kg; for chukar partridges, 9.6 mg/kg; for young coturnix, 4.3 mg/kg; for pigeons (*Columba livia*), 2.0 mg/kg; for prairie sharp-tailed grouse, 2.5 mg/kg; for house sparrows, 3.0 mg/kg; for house finches, 2.1 mg/kg; and for Canada geese, 2.6 mg/kg to dicrotophos when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to dicrotophos was 8,000 ppb (FWPCA, 1968).

Amphibians

The LD_{50} for bullfrogs was 2,000 mg/kg to dicrotophos when the frogs were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Arthrupoda

The 48-hour LC_{50} for stoneflies (*Pteronarcys californica*), waterfleas (*Daphnia pulex*), and amphipods (*Gammarus lacustris*) exposed to dicrotophos was 1,000 ppb, 600 ppb, and 8,000 ppb, respectively (FWPCA, 1968).

The 24-hour LC_{50} for the amphipod (*G. lacustris*) exposed to dicrotophos was 2,300 ppb (Sanders, 1969).

DIELDRIN

Mammals

The LD_{50} for rats was 50 to 55 mg/kg; for rabbits, >150 mg/kg; for dogs, 65 to 95 mg/kg; for young domestic goats, 100 to 200 mg/kg; and for mule deer, 75 to 150 mg/kg to dieldrin when the mammals were given the stated dosages orally (Spector, 1955).

White-tailed deer were fed 5 ppm and 25 ppm of dieldrin daily for up to 3 years (Korschgen and Murphy, 1969). Survival of fawns from treated does was lower in the higher dieldrin dosages than in those from untreated does, with significantly more post-partum mortality. Growth rate of the young females receiving the dieldrin treatment was much slower than that of the untreated females. Some fawns nursing on treated does died, and these fawns had high residues of dieldrin in their brain tissues.

Good and Ware (1969) reported that 5 ppm of dieldrin in the diet of the mouse significantly (0.05) reduced the size of litters from a mean of 9.2 to 8.7.

Sublethal (15 mg/kg/day) dosages of dieldrin were fed to sheep, and this affected the behavior of the sheep (Van Gelder et al., 1969). Dieldrin was found to increase the number of trials required for the animals to relearn a visual discrimination task.

Birds

The LC_{50} for bobwhite quail chicks was 89 ppm and for mallard ducklings, 200 ppm to dieldrin when the birds were fed the stated dosages in their daily diet for 5 days and then fed clean food for 3 days (Heath and Stickel, 1965).

Tucker and Crabtree (1970) computed the LD_{50} for young mallards as 381 mg/kg; for pheasants, 79.0 mg/kg; for young chukar partridges, 23.4 mg/kg; for young coturnix, 69.7 mg/kg; for pigeons (*Columba livia*), 26.6 mg/kg; for house sparrows, 47.6 mg/kg; for Canada geese, 50 to 150 mg/kg; for fulvous tree ducks, 100 to 200 mg/kg; and for young gray partridges, 8.8 mg/kg to dieldrin when the birds were given the stated dosages orally in a capsule.

The LC_{50} for pheasants was 50 to 55 ppm, and for coturnix, 45 to 60 ppm of dieldrin in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

When white king pigeons were fed dieldrin (2 ppm) in their feed for 1 week, steroid metabolism (testosterone increased from 26.7 to 111.4 mμ moles and progesterone increased from 50.1 to 90.5 mμ moles) was significantly increased (Peakall, 1967).

Dieldrin in acetone injected into hen eggs at up to 500 ppm killed only 41 percent of the embryos (Dunachie and Fletcher, 1969); however, chicks which hatched from a 25-ppm dose and were starved were all dead by the 4th day, whereas in untreated controls the mortality was only about 50 percent.

Pheasants were maintained on diets containing various quantities of dieldrin for a 94-day experimental period. In the test, 9 out of 10 females fed 200 ppm per day died within 28 days, and 5 out of 10 females fed 100 ppm died within 58 days. At the close of the experiment, 8 out of 20 females and all 4 males at 50 ppm died; the only male tested at 25 ppm died, whereas the 21 females on this dosage survived (Genelly and Rudd, 1956).

Egg fertility of pheasants fed 50 ppm dieldrin daily was significantly lower than the control (Genelly and Rudd, 1956).

Dieldrin has been reported to cause significant bird kills in areas where it has been applied. For example, after the treatment of fields with dieldrin for the control of a Japanese beetle, large numbers of birds and mammals died, and the bird populations remained at a low level throughout the following spring and summer (Scott, Willis and Ellis, 1959). All resident quail disappeared from a test area treated with 2 pounds of dieldrin per acre (Clawson and Baker, 1959).

Wood pigeons in England were fed under controlled laboratory conditions with dieldrin at dosages of 20, 40, and 80 mg/kg and the toxicities and residues in flesh and various organs of the birds were measured (Turtle et al., 1963). After this investigation wood pigeons in the field were examined for residues. The results of the laboratory and field analyses, indicating dieldrin to be one of the insecticides mainly responsible for wood pigeon deaths, prompted the Ministry of Agriculture, Fisheries and Food to discontinue the use of dieldrin as a seed dressing.

Dieldrin caused a significant (P=from 0.05 to 0.001) decrease in the eggshell thickness of mallard ducks when fed in the diet at 1.6, 4.0, and 10 ppm (Lehner and Egbert, 1969).

In the Netherlands, also, Fuchs (1967) reported numerous deaths in adult wood pigeons during the second half of March, about a week after the peak of the sowing season. In May he made a search for juvenile pigeons and reported finding only about 11 per hectare. More alarming to Fuchs, however, was the large number of birds of prey, buzzards, sparrow hawks (European), and long-eared owls (European) which were found dead. Probably the main source of poisoning for the buzzard was corpses of wood pigeons, and for the sparrow hawk, finches, because they comprise about 18 percent of its diet. The results did not single out any one chlorinated insecticide used as a seed dressing as more dangerous to wood pigeons than another.

Lockie and Ratcliffe (1964) and Lockie, Ratcliffe and Balharry (1969) attributed the decline in breeding success of the golden eagle in west Scotland "mainly to the residues of chlorinated hydrocarbons, particularly dieldrin, in the adult birds and their eggs" (tables 17 A and B).

TABLE 17A. Proportion of eyries of golden eagles having broken eggs during 1937–1963 in west Scotland (Lockie and Ratcliffe, 1964).

Years	Total Eyries Examined (Excluding Those Robbed or Where Birds Not Breeding)	Number of Eyries With Broken Eggs	Percentage of Eyries With Broken Eggs
1937–50	9	1	11
1951–60	26	4	15
1961	6	1	17
1962	7	2	29
1963	9	5	56

TABLE 17B. Breeding success of golden eagles in west Scotland in relation to dieldrin levels in eggs (Lockie, Ratcliffe and Balharry, 1969).

Years	Pct. of Eyries With Young Reared (Number of Eyries With Data)	Mean Dieldrin Level in Eggs, ppm (Number of Eggs Analyzed)
1963–65	31 (29)	0.86 (68)
1966–68	69 (45)	0.34 (78)

Coturnix were fed dieldrin at 5, 10, 50, and 250 ppm in their diets under controlled conditions (Stickel, Stickel and Spann, 1969). When half of the birds at each dosage had died, the other half were killed for comparison of dieldrin residues. Although the range of residues in the dead and survivors overlapped somewhat, brain residues correlated well with the death of the birds and coincided with data from the field. The authors concluded there are species differences, but that brain residues of 4 or 5 ppm (wet weight) or higher were hazardous and would implicate dieldrin as a prime suspect of the cause of death.

Dieldrin was fed to hen pheasants through 2 generations (Baxter, Linder and Dahlgren, 1969). First-generation hens were given 4 and 6 mg of dieldrin per week for 15 weeks with no mortality; the offspring of these hens received 6 mg per week for a total of 14 weeks. Some mortality (25 to 50 percent) occurred in both of the second-generation groups. Fertility and hatchability of eggs were significantly lower in the second-generation hens. Chick survival and growth appeared normal. However, behavior of chicks from hens receiving 6 mg dieldrin for 14 weeks was affected; these chicks tended to choose the deep side of a visual cliff.

Mallard ducks were given dieldrin in their feed for 16 months, and eggs were collected during months 2, 3, 4, 14, 15, and 16 (periods coinciding with waterfowl nesting in the wild). These eggs had shells averaging 0.0085 mm thinner (at 1.6 ppm dosage, $P=0.001$), 0.0066 mm thinner (at 4 ppm dosage, $0.02<P<0.01$), and 0.0185 mm thinner (at 10 ppm dosage, $P=0.001$) than the untreated control (Lehner and Egbert, 1969). Herring gulls were also found to have 4-percent thinner eggshells at about 44 ppm of dieldrin residue in the egg yolks (unpublished results of P.N.L. in Lehner and Egbert, 1969). However, dieldrin fed to hen pheasants in capsules at dosages of 4, 6, or 10 mg did not cause any significant thinning of the eggshell (Dahlgren and Linder, 1970).

Fishes

The LC_{50} of dieldrin tested against various species of fish is found in table 18.

The 24-hour LC_{50} for rainbow trout exposed to dieldrin at temperatures of 1.6°C, 7.2°C, and 12.7°C was 13 ppb, 5.1 ppb, and 3.1 ppb, respectively (Macek, Hutchinson and Cope, 1969); and the 24-hour LC_{50} for bluegills exposed at temperatures of 12.7°C, 18.3°C, and 23.9°C was 30 ppb, 24 ppb, and 15 ppb, respectively.

The relative toxicity of dieldrin to 3 species of fishes as measured by the 48-hour EC_{50} was as follows: rainbow trout at 5 ppb, 13°C; bluegill at 8 ppb, 24°C; and channel catfish at 25 ppb, 24°C (Cope, 1966).

As both temperature and time increase, the LC_{50} to small (about 1 gram) bluegills decreases (table 19).

Dieldrin pellets disseminated over 2,000 acres at a rate of 1 lb/A for sandfly (*Culicoides*) larval control severely affected the biological community in the Florida East Coast tidal marsh (Harrington and Bidlingmayer, 1958). The treatment killed an estimated 20 to 30 tons of fish of 30 species, including many sport fish, such as young tarpon, which utilize the salt marshes as nursing grounds ([illegible], 1958).

TABLE 18. The LC_{50} for various fish to dieldrin.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Bluegill	24	0.0055	Cope, 1965
Rainbow trout	24	0.05	Mayhew, 1955
Rainbow trout	24	0.010	Cope, 1965
Harlequin fish	24	0.24	Alabaster, 1969
Bluegill	48	0.0024	FWPCA, 1968
Bluegill	96	0.008	Henderson, Pickering and Tarzwell, 1959
Goldfish	96	0.037	"

TABLE 19. Effects of increasing temperature and exposure time on the toxicity of dieldrin to bluegills (Cope, 1965).

Temperature, °F	LC_{50} (ppm)		
	24 hrs	48 hrs	96 hrs
45	0.054	0.034	0.018
55	0.040	0.026	0.018
65	0.024	0.018	0.0145
75	0.014	0.011	0.0092
85	0.010	0.0084	0.0071

The toxicity to dieldrin in three species of fish collected in the field at Twin Bayou, Mississippi, where the populations had been exposed to heavy concentrations of several insecticides used in the adjoining cotton acreages, compared with a control population, as measured by 36-hour LC_{50}, were: golden shiner, control 25 ppb versus Twin Bayou 900 ppb; bluegills, control 25 ppb versus Twin Bayou 900 ppb; and green sunfish, control 33 ppb versus Twin Bayou 1,250 ppb (Ferguson et al., 1965b). In another investigation the toxicity to dieldrin in resistant mosquito fish and black bullheads collected from streams in Mississippi compared with that in an unexposed control population, as measured by 36-hour LC_{50}, were: mosquito fish, control 16 ppb versus resistant (Sidon, Miss.) 800 ppb; and black bullhead, control 2.5 ppb versus resistant (Wayside, Miss.) 55 ppb (Ferguson et al., 1966a).

Growth rates of rainbow trout were reduced by dieldrin concentrations above only 0.12 ppb (Chadwick and Shumway, 1969). Eggs (embryos), however, exposed to dieldrin concentrations as high as 52 ppm survived well.

An investigation of the persistence of dieldrin in fish revealed that 50 percent of the chemical was lost in about 1 month (Macek, 1969).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles and chorus frogs exposed to dieldrin was 1.1 ppm and 0.20 ppm, respectively (Sanders, 1970).

Molluscs

Molluscs were apparently unharmed after an application of dieldrin at 1.0 lb/A to a coastal tidal marsh in Florida (Harrington and Bidlingmayer, 1958).

Arthropods and Annelids

The LC_{50} for various arthropods to dieldrin is found in table 20.

TABLE 20. The LC_{50} for various arthropods to dieldrin.

Arthropod Species	Exposure (hours)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcella badia*)	96	0.0005	Sanders and Cope, 1968
" (*Claassenia sabulosa*)	96	0.00058	"
" (*Pteronarcys californica*)	96	0.039	"
Sand shrimp	96	0.007	Eisler, 1969
Hermit crab	96	0.018	"
Grass shrimp	96	0.050	"
Amphipod (*Gammarus lacustris*)	96	>0.107	"
Stonefly (*P. californica*)	24	1.4	Sanders, 1969
" (*P. californica*)	48	0.0013	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	0.100	Sanders and Cope, 1966
" (*D. pulex*)	48	0.240	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.250	Sanders and Cope, 1966
	48	1.000	FWPCA, 1968

The 48-hour EC_{50} (immobilization value at 60° F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to dieldrin was 240 ppb and 250 ppb, respectively (Sanders and Cope, 1966).

The relative toxicity of dieldrin to 4 species of invertebrates, as measured by the 48-hour EC_{50}, was as follows: stonefly nymph (*P. californicus* [*sic*]) at 16 ppb, mayfly nymph (*Baetis* sp.) at 19 ppb, waterflea (*S. serrulatus*) at 9 ppb, and waterflea (*D. pulex*) at 0.4 ppb (Cope, 1966).

Dieldrin distributed at 1.0 lb/A in Florida marshes completely annihilated the crab population and virtually exterminated the crustaceans (Harrington and Bidlingmayer, 1958).

When about 18,000 acres of Illinois farmland were treated with 2 or 3 lb/A of dieldrin for a 5-year period for Japanese beetle control, Luckmann (1960) found that the European corn borer population in the area increased 160 percent. The corn borer outbreak was presumably the result of the destruction of some of the natural enemies of the corn borer.

Luckmann and Decker (1960) treated fields in Illinois with 2 and 3 lb/A of dieldrin and found that dryinids, common parasites of cicadellids and fulgorids, were clearly absent in treated regions. Predaceous carabids, particularly larvae, were noticeably absent for 2 years and few larvae were present during the third year. There were no differences, however, in the numbers of other insects and earthworms. Doane (1962) reported that dieldrin applied at a rate of 10 lb/A for insect control eliminated most of the earthworms from the treated plots for 18 months after treatment.

Plants

Exposing phytoplankton communities for 4 hours to 1 ppm of dieldrin reduced the productivity of these communities 84.8 percent (Butler, 1963a).

Dieldrin applied to soils at 5 lb/A to a depth of 5 inches was found to be translocated into cucumbers growing on the soil (Lichtenstein et al., 1965).

Microorganisms

Jones (1956) reported that dieldrin was considerably more toxic to the bacteria converting ammonia to nitrates in the soil than bacteria changing organic matter to ammonia. Dieldrin was far more toxic to soil bacteria than were such other chlorinated insecticides as DDT, BHC, endrin, and methoxychlor.

When dieldrin was applied to water at 12.8 ppm, the number of diatoms (*Navicula seminulum* var. *Hustedtii*) produced was reduced by 50 percent (Cairns, 1968). At 32.0 ppm of dieldrin in water all diatom production was halted.

Biological Concentration

Trout concentrated the level of dieldrin 3,300-fold in their bodies when the water contained 0.0023 ppm of dieldrin (Holden, 1966).

Earthworms collected from 2 Missouri fields treated with aldrin for at least 15 of the past 17 years contained 4.0 ppm (dry weight) of dieldrin (Korschgen, 1970). The levels in the earthworms were 11 times those found in the soil. In this same habitat in August ground beetles were found to contain 16 to 22 ppm of dieldrin, or about 40 times the soil level. Both earthworms and ground beetles serve as food for birds and other animals.

After the treatment of plots with dieldrin at ½, 2, and 8 lb/A, periodic analyses were made of the earthworms in the plots (USDI, 1967). Three days after treatment the residues in the earthworms were 4.8, 9.7, and 14.6 ppm; by day 340 the residues had declined to 1.0, 2.4, and 4.7 ppm. The report concluded: "It is clear that extremely dangerous levels prevailed in the worms for many months."

Eastern oysters exposed in flowing seawater for 10 days to dieldrin at 0.001 ppm concentrated the toxicant 1,000 times (1 ppm) (Wilson, 1965).

Analysis at harvest time showed that peanuts concentrated dieldrin from soil at a level of 0.14 ppm to 0.75 ppm in peanut meat (Beck et al., 1962).

Four species of algae concentrated dieldrin about 150-fold when they were exposed to 1 ppm in water for 7 days (Vance and Drummond, 1969).

Persistence

Dieldrin applied at 100 ppm persisted in soil for >6 years (Westlake and San Antonio, 1960).

Dieldrin in soil persisted for >9 years (Wilkinson, Finlayson and Morley, 1964).

Dieldrin applied at 25 ppm to soil persisted (50 percent loss) for about 8 years, and dieldrin remaining 15 years after application at a rate of 100 ppm to sandy loam soil was 31 percent (Nash and Woolson, 1967).

DILAN

Mammals

The LD_{50} for rats was 475 to 600 mg/kg to Dilan when the mammals were fed the stated dosage orally (FCH, 1970).

Fishes

The 48-hour LC_{50} for bluegill exposed to Dilan was 16 ppb (FWPCA, 1968).

Arthropods

The 24-hour LC_{50} for an amphipod (*Gammarus lacustris*) exposed to Dilan was 800 ppb (Sanders, 1969).

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) and amphipods (*G. lacustris*) exposed to Dilan was 21 ppb and 600 ppb, respectively (FWPCA, 1968).

Persistence

Dilan applied at 50 ppm to soil persisted (50 percent loss) for 4 years, and Dilan remaining 14 years after application at a rate of 100 ppm to sandy loam soil was 25 percent (Nash and Woolson, 1967).

DIMANIN

Fishes

The 24-hour LC_{50} for harlequin fish to dimanin was 1.3 ppm (Alabaster, 1969).

DIMETHOATE

Mammals

The LD_{50} for the rat was 185 to 245 mg/kg (Neumeyer, Gibbons and Trask, 1969) and for the mule deer, ≈200 mg/kg (Tucker and Crabtree, 1970) to dimethoate when the animals were given the stated dosages orally in a capsule.

Red clover fields in Indiana were treated with dimethoate at ¼ and ½ lb/A (Barrett and Darnell, 1967). After the treatments the mouse (house) population decreased by 50 percent at ¼ lb/A and decreased by 80 percent at ½ lb/A; the prairie deer mouse population remained unchanged, and the prairie vole population increased by 5 times at ¼ lb/A and increased by 4 times at ½ lb/A. Because of the movement of all species of mice, changes in the mouse populations were also observed in the control plots. The authors postulated that the abrupt loss of insect prey due to the insecticide caused the mouse populations to change.

Birds

The LD_{50} for young mallards was 41.7 mg/kg to dimethoate when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was 900 to 1,100 ppm; for pheasants, 300 to 400 ppm; and for coturnix, 300 to 400 ppm of dimethoate in the diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Fishes

The LC_{50} for bluegills to dimethoate was reported by Cope (1965) to be 28 ppm for a 24-hour exposure, and the 24-hour LC_{50} for rainbow trout to dimethoate was 19 ppm (Alabaster, 1969). The 48-hour LC_{50} for bluegill exposed to dimethoate was 9,600 ppb (FWPCA, 1968).

Arthropods

The LC_{50} for various arthropods to dimethoate is found in table 21.

The treatment of fields of red clover in Indiana with dimethoate at ¼ and ½ lb/A resulted in significant reductions in populations of Orthoptera, Hemiptera, and Homoptera and only slight reductions in populations of Lepidoptera, Coleoptera, Diptera, and Hymenoptera (Barrett and Darnell, 1967).

Persistence

Dimethoate in soil persisted for <9 months (Mulla, Georghiou and Cramer, 1961).

TABLE 21. The LC_{50} for various arthropods to dimethoate

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcys californica*)	24	0.510	Sanders and Cope, 1969
Amphipod (*Gammarus lacustris*)	24	0.900	Sanders, 1969
Stonefly (*P. californica*)	48	0.140	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.400	"
Red crawfish	48	>1	Muncy and Oliver, 1963
Waterflea (*Daphnia magna*)	48	2.5	FWPCA, 1968
Stonefly (*Pteronarcys* sp.)	48	0.140	Cope, 1965

DIMETHRIN

Fishes

The 48-hour LC_{50} for rainbow trout exposed to dimethrin was 700 ppb (FWPCA, 1968).

DIOTHYL

Fishes

The 24-hour LC_{50} for harlequin fish to diothyl was 5.2 ppm (Alabaster, 1969).

DIOXATHION

Mammals

The LD_{50} for male rats was 110 mg/kg to dioxathion when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

The LC_{50} for mallards was 3,500 to 4,700 mg/kg; for pheasants, 4,000 to 4,400 mg/kg; and for coturnix, >5,000 mg/kg to dioxathion when the birds were fed the toxicant in feed for 5 days plus 3 days of clean feed (Heath et al., 1970a).

When chickens were fed dioxathion at a dosage of 320 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The 48-hour LC_{50} for bluegill exposed to dioxathion was 14 ppb (FWPCA, 1968).

Arthropods and Annelids

The 48-hour LC_{50} for amphipods (*Gammarus lacustris*) exposed to dioxathion was 690 ppb (FWPCA, 1968).

The LC_{50} for sand shrimp, grass shrimp, and hermit crab to dioxathion for a 24-hour exposure was 307 ppb, 500 ppb, and 300 ppb, respectively (Eisler, 1969).

The 96-hour LC_{50} for an amphipod (*G. lacustris*) exposed to dioxathion was 880 ppb (Sanders, 1969).

DISULFOTON

Mammals

The LD_{50} for rats was 12.5 mg/kg (FCH, 1970), and for domestic goats, < 15.0 mg/kg (Tucker and Crabtree, 1970) to disulfoton when the mammals were fed the stated dosages orally.

Birds

The LD_{50} for young mallards was 6.5 mg/kg to disulfoton when the birds were fed the stated dosage orally (Tucker and Crabtree, 1970). The LC_{50} for mallards was 400 to 600 ppm; for pheasants, 600 to 700 ppm; for bobwhites, 700 to 800 ppm; and for coturnix, 300 to 400 ppm of disulfoton in diets of 2-week-old birds when fed treated for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

When chickens were fed disulfoton at a dosage of 32 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The 48-hour LC_{50} for bluegill exposed to disulfoton was 40 ppb (FWPCA, 1968).

Arthropods

The estimated 24-hour LC_{50} for stonefly nymphs (*P. californica*) to disulfoton was 10 ppb (Sanders and Cope, 1968).

The 48-hour LC_{50} for stoneflies (*P. californica*) and amphipods (*Gammarus lacustris*) exposed to disulfoton was 18 ppm and 70 ppm, respectively (FWPCA, 1968).

The 96-hour LC_{50} for an amphipod (*G. lacustris*) exposed to disulfoton was 110 ppb (Sanders, 1969).

Persistence

Disulfoton applied to soil persisted for about 4 weeks (Kearney, Nash and Isensee, 1969).

DN-111

Fishes

In field applications of DN-111 at 1 to 10 ppm most fish were killed in the ponds (Kuntz and Wells, 1951 in Springer, 1957).

Molluscs and Arthropods

Field applications of DN-111 at 1 to 10 ppm drastically reduced snails, but the arthropods were not significantly reduced (Kuntz and Wells, 1951 in Springer, 1957).

DN-111 applied, as it is in orchards, at a rate of 0.20 lb/100 gal of water caused some mortality in some beneficial predatory coccinellid beetles (especially *Stethorus picipes*), but caused little or no mortality to beneficial parasitic wasps (Bartlett, 1963).

DNOC

Mammals

The LD_{50} for the rat was 26 to 30 mg/kg to DNOC when the mammal was fed the stated dosage orally (Spector, 1955).

Applications of DNOC for weed control in crops at rates of 1 to 6 lb/A were reported to have killed some rabbits (*Oryctolagus cuniculus*) in England, mainly through the ingestion of contaminated food (Edson, 1954 in Springer, 1957).

The LD_{50} for the rat was 10 to 50 mg/kg to DNOC when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The lethal doses of DNOC to pheasants was: DNOC (ammonium salt) at 80 mg/kg and DNOC (sodium salt) at 25 mg/kg (Paludan, 1953 in Springer, 1957).

The LD_{50} for young mallards was 22.7 mg/kg to DNOC when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

DNOC in acetone injected into hen eggs at 5 ppm, 10 ppm, and 25 ppm killed 25, 75, and 100 percent of the embryos (Dunachie and Fletcher, 1969).

Dambach and Leedy (1949) reported that the dinitrophenols were repellent to birds. Some pheasants and song birds were poisoned when ingesting food from crop areas treated with 1 to 6 lb/A of DNOC (Edson, 1954 in Springer, 1957).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to DNOC was 210 ppb (FWPCA, 1968). Szumlewicz and Kemp (1951 in Springer, 1957) reported also that in the laboratory DNOC (sodium salt), killed all the fish at 1 ppm.

Molluscs

DNOC (sodium salt) at 1 ppm killed 100 percent of the snails in a laboratory experiment (Szumlewicz and Kemp, 1951 in Springer, 1957).

Arthropods and Annelids

The estimated 24-hour LC_{50} for stonefly nymphs (*Pteronarcys californica*) to DNOC was 0.32 ppm (Sanders and Cope, 1968).

The 48-hour LC_{50} for stoneflies (*P. californica*) exposed to DNOC was 560 ppb (FWPCA, 1968).

DNOC applied at 5.6 lb/A had no effect on *Allolobophora caliginosa*, but caused 52 percent mortality in *Lumbricus castaneus* (earthworms) (Van der Drift, 1963).

Persistence

DNOC applied at 50 ppm persisted in soil for 7 days (Bruinsma, 1960).

DURSBAN

Mammals

The LD_{50} for the rat was 135 mg/kg (Neumeyer, Gibbons and Trask, 1969), and for domestic goats, 500 to 1,000 mg/kg (Tucker and Crabtree, 1970) to dursban when the mammals were given the stated dosages orally in a capsule.

Birds

The LD_{50} for mallards was 70 to 80 mg/kg; for young pheasants, 8.4 to 17.7 mg/kg; for young chukar partridges, about 61 mg/kg; for young coturnix, 13 to 18 mg/kg; for pigeons (*Columba livia*), 26.9 mg/kg; for house sparrows, 21.0 mg/kg; for Canada geese, ≈80 mg/kg; and for lesser sandhill cranes, 25 to 50 mg/kg to dursban when the birds were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970). The LC_{50} for coturnix was 275 to 300 ppm of dursban in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Dursban applied at 0.10 lb/A had no observable effect on mallards and pheasants (Burgoyne, 1968).

When chickens were fed dursban at a dosage of 200 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The 24-hour LC_{50} for rainbow trout exposed to dursban at temperatures of 1.6°C, 7.2°C, and 12.7°C was 550 ppb, 110 ppb, and 58 ppb, respectively (Macek, Hutchinson and Cope, 1969).

The 48-hour LC_{50} for rainbow trout exposed to dursban was 20 ppb (FWPCA, 1968).

Three species of fish were collected in the field in the Mississippi Delta area where the populations had been exposed to heavy concentrations of chlorinated insecticides plus parathion from the treated cotton acreages (Ferguson, Gardner and Lindley, 1966). Although these fish had never been exposed to dursban, the toxicity in these populations to dursban compared with that of a control population as measured by 36-hour LC_{50} were: golden shiners, control 45 ppb versus resistant 125 ppb; mosquito fish, control 230 ppb versus resistant 595 ppb; and green sunfish, control 37.5 ppb versus resistant 125 ppb.

Dursban applied at 0.10 lb/A had no effect on brown bullheads in ponds on a refuge in California (Burgoyne, 1966).

An investigation of the persistence of dursban in fish revealed that about 50 percent of the chemical was lost in ≤1 week (Smith, Watson and Fischer, 1966).

Arthropods

The LC_{50} for various arthropods to dursban is found in table 22.

Rice fields in California treated with dursban at 0.0125 and 0.025 lb/A caused little or no mortality to non-target insect species, Corixidae, *Belostoma* sp., *Tropisternus lat. humeralis*, *Laccophilus* sp., and Hydrophilidae (Washino, Whitesell and Womeldorf, 1968).

TABLE 22. The LC_{50} for various arthropods to dursban.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	0.00076	Sanders, 1969
Stonefly (*Pteronarcella badia*)	24	0.0043	Sanders and Cope, 1968
" (*Claassenia sabulosa*)	24	0.0092	"
" (*Pteronarcys californica*)	24	0.050	"
Amphipod (*G. lacustris*)	48	0.0004	FWPCA, 1968
Stonefly (*P. badia*)	48	0.0019	"

ENDOSULFAN

Mammals

The LD_{50} for the rat was 100 mg/kg to endosulfan when the mammal was fed the stated dosage orally (USDI, 1970).

Birds

Tucker and Crabtree (1970) reported the LD_{50} for young mallards as 33.0 mg/kg to endosulfan when the birds were fed the stated dosage orally in capsules. The LC_{50} for mallards was 900 to 1,100 ppm; for pheasants, 1,200 to 1,350 ppm; for bobwhites, 800 to 900 ppm; and for coturnix, 2,100 to 2,250 ppm of endosulfan in the diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

Endosulfan in acetone injected into hen eggs at 5 ppm, 100 ppm, 200 ppm, 300 ppm, 400 ppm, and 500 ppm killed 19, 46, 40, 47, 31, and 47 percent of the embryos (Dunachie and Fletcher, 1969). The authors could not explain the inconsistency.

Fishes

The 24-hour LC_{50} for rainbow trout exposed to endosulfan at temperatures of 1.6°C, 7.2°C, and 12.7°C was 18 ppb, 6.1 ppb, and 3.9 ppb, respectively (Macek, Hutchinson and Cope, 1969).

The 48-hour LC_{50} for rainbow trout exposed to endosulfan was 1.9 ppb (FWPCA, 1968).

The 24-hour LC_{50} for harlequin fish to endosulfan was 0.09 ppb (Alabaster, 1969).

Arthropods

The LC_{50} for various arthropods to endosulfan is found in table 23.

TABLE 23. The LC_{50} for various arthropods to endosulfan.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	0.0062	Sanders, 1969
Stonefly (*Pteronarcys californica*)	24	0.024	Sanders and Cope, 1968
" (*P. californica*)	48	0.0056	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.064	"
Waterflea (*Daphnia pulex*)	48	0.240	"

ENDRIN

Mammals

The LD_{50} for rats was ≤5 to 43 mg/kg; for rabbits, 5 to 10 mg/kg; for guinea pigs, 10 to 36 mg/kg (Negherbon, 1959); and for domestic goats, 25 to 50 mg/kg (Tucker and Crabtree, 1970) to endrin when the mammals were given the stated dosages in a capsule.

Good and Ware (1969) reported that 5 ppm of endrin in the diet of the mouse significantly (0.05) reduced the size of litters from a mean of 9.2 to [illegible].

Endrin at 1.2 to 1.4 lb/A was found to eliminate most wild mice in orchards (Wolfe, 1957).

Wild pine mice exposed to [illegible] in their natural habitat were found to have [illegible] times greater tolerance to the insecticide than normal unexposed mice (Webb and Horsfall, 1967). Although most of the resistance was believed to be genetic, a certain amount of tolerance could be conferred by feeding the mice sublethal dosages of endrin.

Birds

Tucker and Crabtree (1970) computed the LD_{50} for mallards as 5.6 mg/kg; for young pheasants, 1.8 mg/kg; and for pigeons (*Columba livia*), 2.0 to 5.0 mg/kg to endrin when the birds were fed the stated dosages orally in capsules.

Endrin in acetone injected into hen eggs at 25 ppm, 50 ppm, and 100 ppm killed 77, 61, and 70 percent of the embryos (Dunachie and Fletcher, 1969). The authors noted the inconsistent results. Chicks which hatched from a 5-ppm dose and starved were all dead by the 5th day, whereas in untreated controls the mortality was only about 50 percent.

The LC_{50} for coturnix was 15 to 18 ppm of endrin in diets of 2-week-old birds when fed treated feed for 5 days followed by clean food for 3 days. The LC_{50} for bobwhite quail chicks was 15 ppm; for pheasant chicks, 11 ppm; and for mallard ducklings, 21 ppm to endrin when the birds were fed the stated dosages daily in the food for 5 days and then fed clean food for 3 days (Heath and Stickel, 1965).

Only an occasional quail or pheasant was killed when orchard ground cover was treated with 1.2 to 1.4 lb/A of endrin (Wolfe, 1957).

Fishes

The LC_{50} for endrin tested against various species of fish is found in table 24.

The 24-hour LC_{50} for rainbow trout exposed to endrin at temperatures of 1.6°C, 7.2°C, and 12.7°C was 15 ppb, 5.8 ppb, and 2.8 ppb, respectively (Macek, Hutchinson and Cope, 1969); and the 24-hour LC_{50} for bluegills exposed at temperatures of 12.7°C, 18.3°C, and 23.8°C was 2.8 ppb, 1.5 ppb, and 0.6 ppb, respectively.

TABLE 24. The LC_{50} for various fish to endrin.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Bluegill	24	0.00035	Cope, 1965
Rainbow trout	24	0.0018	"
Carp	48	0.14	Iatomi et al., 1958
Bluegill	48	0.2	FWPCA, 1968
Bluntnose minnow	96	0.0002	Katz and Chadwick, 1961
Bluegill	96	0.0006	Henderson, Pickering and Tarzwell, 1959
Fathead minnow	96	0.0015	"
Northern puffer	96	0.0031	Eisler and Edmunds, 1966

The toxicity of endrin to 3 fishes as measured by 48-hour EC_{50} was as follows: bluegill at 0.6 ppb, 24°C; rainbow trout at 0.6 ppb, 13°C; and channel catfish at 1 ppb, 24°C (Cope, 1966).

Low concentrations of endrin (0.5 ppb) prevented reproduction in guppies; endrin was also observed to cause increased activity in guppies at the low dosages, possibly interrupting normal swarming and displacement behavior (Mount, 1962).

Adult northern puffers (a marine fish) were found to survive endrin at a concentration of 1 ppb for 24 and 96 hours, but succumbed at 10 ppb at 24 hours (Eisler and Edmunds, 1966).

As both time of exposure and temperature increased, the LC_{50} for both rainbow trout and bluegills decreased (table 25).

Three species of fish were collected in the field at Twin Bayou, Mississippi, where the populations had been exposed to heavy concentrations of several insecticides used in the adjoining cotton acreage (Ferguson et al., 1965b). The toxicity of endrin in these fish compared with that in a control population, as measured by 36-hour LC_{50}, were: golden shiner, control 8.0 ppb versus Twin Bayou 310 ppb; bluegills, control 1.5 ppb versus Twin Bayou 300 ppb; and green sunfish, control 3.4 ppb versus Twin Bayou 160 ppb. In another investigation resistant mosquito fish and black bullheads were collected from streams in Mississippi (Ferguson et al., 1965a). The toxicity to endrin in these fish compared with that in an unexposed control population, as measured by 36-hour LC_{50}, were: mosquito fish, control 1 ppb versus resistant (Sidon, Miss.) 180 ppb; and black bullhead, control 0.87 ppb versus resistant (Wayside, Miss.) 2.5 ppb.

TABLE 25. The effects of increasing the exposure time and temperature on the toxicity of endrin to small (approximately 1 g) rainbow trout and bluegills (Cope, 1966).

Temperature (°F)	Fish	LC_{50} (ppb)		
		24 hrs	48 hrs	96 hrs
45	Rainbow trout	14.5	6.5	2.4
"	Bluegill	6.2	1.6	0.7
55	Rainbow trout	5.2	2.4	1.6
"	Bluegill	3.3	1.4	0.7
65	Rainbow trout	2.5	1.9	1.1
"	Bluegill	1.4	0.7	0.4
75	Rainbow trout	1.5	1.3	0.75
"	Bluegill	0.8	0.6	0.4
85	Bluegill	0.3	0.2	0.2

The margin of safety and lethality for fish exposed to endrin was found to be extremely narrow (Lowe, 1966). Juvenile spot exposed to 0.05 ppb of endrin for 5 weeks were not affected as measured by mortality, growth rate, histology, and stress; however, increasing the dosage to only 0.1 ppb killed the fish in 5 days.

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles and chorus frog tadpoles exposed to endrin was 0.27 ppm and 0.20 ppm, respectively (Sanders, 1970).

Arthropods and Annelids

The LC_{50} for various arthropods to endrin is found in table 26.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to endrin was 26 ppb and 20 ppb, respectively (Sanders and Cope, 1966).

The toxicity of endrin to 4 invertebrates, as measured by the 48-hour EC_{50}, was as follows: waterflea (*S. serrulatus*) at 26 ppb, waterflea (*D. pulex*) at 20 ppb, mayfly nymph (*Baetis* sp.) at 5 ppb, and stonefly nymph (*P. californicus [sic]*) at 1 ppb (Cope, 1966).

The LC_{50} of endrin for a 48-hour exposure against red crawfish was 0.3 ppm (Muncy and Oliver, 1963).

Hopkins and Kirk (1957) reported no mortality from endrin at 5 lb/A when tested against the manure worm, *Eisenia* sp., in laboratory experiments.

Azuki-bean weevil adults which survived endrin treatments produced 22 percent fewer eggs than unexposed weevils (Kiyoko and Tamaki, 1969).

Plants

Productivity in phytoplankton exposed for 4 hours to 1 ppm of endrin was reduced 46 percent (Butler, 1963a).

Endrin at dosages of 10 and 100 ppm in soil significantly reduced bean growth (12.4 g at 100 ppm) compared with the control (16.2 g) at the end of 8 weeks of exposure (Cole et al., 1968). At dosages of 1, 10, and 100 ppm in soil endrin also caused significant changes in the macro and micro

TABLE 20. The LC_{50} for various arthropods to endrin.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcella badia*)	96	0.00054	Sanders and Cope, 1968
Sand shrimp	96	0.0017	Eisler, 1969
Stonefly (*Claassenia sabulosa*)	96	0.00076	Sanders and Cope, 1968
" (*Pteronarcys californica*)	96	0.004	"
Amphipod (*Gammarus lacustris*)	96	0.0030	Sanders, 1969
Grass shrimp	96	0.0018	Eisler, 1969
Hermit crab	96	0.012	"
Stonefly (*P. californica*)	48	0.0008	FWPCA, 1968
" (*P. californica*)	48	0.00032	Sanders and Cope, 1966
Amphipod (*G. lacustris*)	48	0.0047	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	0.020	Sanders and Cope, 1966
" (*D. pulex*)	48	0.020	FWPCA, 1968

element (N, P, K, Ca, Mg, Mn, Fe, Cu, B, Al, Sr, and Zn) constituents of above-ground portions of both corn and bean plants. For example, iron increased in the endrin (10 ppm)-treated plants (308 ppm dry weight) compared with the control (288 ppm); however, at this same dosage copper decreased (8.2 ppm) compared with the control (10.5 ppm).

Biological Concentration

Fathead minnows exposed to water containing 0.015 ppb of endrin concentrated the endrin in their own bodies by 10,000 times (Mount and Putnicki, 1966).

Eastern oysters were observed to concentrate endrin from water (Butler, 1964). Wilson (1965) reported that oysters exposed to endrin at 0.001 ppm concentrated the endrin about 1,000 times during a 10-day exposure period.

Following the 7-day exposure of 4 algae species at 1 ppm of endrin, the algae concentrated endrin by about 170-fold under the test conditions (Vance and Drummond, 1969).

Persistence

Endrin in soil persisted for >9 months (Mulla, 1960).

Endrin applied at 25 ppm to soil persisted (50-percent loss) for 12 years, and endrin remaining 14 years after application at a rate of 100 ppm to sandy loam soil was 41 percent (Nash and Woolson, 1967).

EPH

Fishes

The 48-hour LC_{50} for bluegill exposed to EPH was 17 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) and amphipods (*Gammarus lacustris*) exposed to EPH was 0.1 ppb and 38 ppb, respectively (FWPCA, 1968).

EPN

Mammals

The LD_{50} for the rat was 14 to 42 mg/kg to EPN when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for young mallards was 3.1 mg/kg; for young pheasants, 53.4 mg/kg; for young chukar partridges, 14.5 mg/kg; for young coturnix, 5.3 mg/kg; and for pigeons (*Columba livia*), 3.9 mg/kg to EPN when the birds were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970). The LC_{50} for pheasants was 550 to 1,150 ppm; for bobwhites, 500 to 550 ppm; and for coturnix, 250 to 300 ppm of EPN in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

When chickens were fed EPN at a dosage of 40 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Arthropods

The 24-hour LC_{50} for an amphipod (*Gammarus lacustris*) exposed to EPN was 100 ppb (Sanders, 1969).

Persistence

EPN applied in soil persisted for <5 years (Terriere and Ingalsbe, 1955).

ETHION

Mammals

The LD_{50} for rats was 96 mg/kg to ethion when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

Ethion in acetone injected into hen eggs at 200 ppm and 300 ppm killed 24 and 36 percent of the embryos (Dunachie and Fletcher, 1969). This toxicant also caused teratogenic effects, especially when combined with malathion in a 1 to 5 combination.

When chickens were fed ethion at a dosage of 400 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The 48-hour LC_{50} for bluegill exposed to ethion was 230 ppb (FWPCA, 1968).

The 24-hour LC_{50} for harlequin fish to ethion was 0.7 ppm (Alabaster, 1969).

Arthropods

The LC_{50} for various arthropods to ethion is found in table 27.

TABLE 27. The LC_{50} for various arthropods to ethion

Arthropod Species	Exposure (hours)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	0.0056	Sanders, 1969
Stonefly (*Pteronarcys californica*)	96	0.026	Sanders and Cope, 1968
Waterflea (*Daphnia magna*)	48	0.00001	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.0032	"
Stonefly (*P. californica*)	48	0.014	"

FENITROTHION

Mammals

The LD_{50} for rats was 250 mg/kg (FCH, 1970) and for the mule deer, 727 mg/kg (Tucker and Crabtree, 1970) to fenitrothion when the animals were given the stated dosage orally in a capsule.

Birds

The LD_{50} for young bobwhite quail was 47.4 mg/kg to fenitrothion when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

The LC_{50} for pheasants was 450 to 500 ppm to fenitrothion in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

Persistence

One year after the treatment of northwestern Ontario forest with fenitrothion at 6 oz/A and phosphamidon at 4 oz/A the long-term effects were evaluated on predaceous carabid beetles and lycosid spiders (Freitag and Poulter, 1970). The populations of these predators were clearly suppressed in the treated area. The authors stated that the results did "not imply a 1 year persistence of the insecticides, but rather a persistent disturbance of the ecosystem."

FENSULFOTHION

Mammals

The LD_{50} for the rat was 2 to 10 mg/kg to fensulfothion when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for young mallards was 0.75 mg/kg to fensulfothion when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was 40 to 50 ppm; for pheasants, 140 to 180 ppm; and for bobwhites, 30 to 40 ppm of fensulfothion in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

FENTHION

Mammals

The LD_{50} for rats was about 200 to 300 mg/kg to fenthion when the animals were fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for young mallards was 5.9 mg/kg; for pheasants, 17.8 mg/kg; for young chukar partridges, 25.0 mg/kg; for young coturnix, 10.6 mg/kg; for pigeons (*Columba livia*), 4.6 mg/kg; for mourning doves, 2.7 mg/kg; for house sparrows, 23.7 mg/kg; for house finches, ~10 mg/kg; and for Canada geese, 19.0 mg/kg to fenthion when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was 200 to 230 ppm; for pheasants, 180 to 220 ppm; for bobwhites, 27 to 33 ppm; and for coturnix, 80 to 90 ppm of fenthion in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

When chickens were fed fenthion at a dosage of 25 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fenthion applied at 0.01 lb/A had no observable effect on mallards and pheasants in a wildlife refuge in California (Burgoyne, 1968).

Fishes

Application of fenthion at 0.01 lb/A to the California refuge had no effect on brown bullheads (Burgoyne, 1968).

The LC_{50} for various fish to fenthion is found in table 28.

Arthropods

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to fenthion, was 0.62 ppb and 0.80 ppb, respectively (Sanders and Cope, 1966).

The LC_{50} for various arthropods to fenthion is found in table 29.

TABLE 28. The LC_{50} for various fish to fenthion

Fish Species	Exposure (hours)	LC_{50} (ppm)	Source
Brown trout	48	0.090	FWPCA, 1968
Rainbow trout	96	0.930	Macek and McAllister, 1970
Carp	96	1.160	"
Coho salmon	96	1.320	"
Brown trout	96	1.330	"
Bluegill	96	1.380	"
Largemouth bass	96	1.540	"
Black bullhead	96	1.620	"
Yellow perch	96	1.650	"
Channel catfish	96	1.680	"
Redear sunfish	96	1.880	"
Fathead minnow	96	2.440	"
Goldfish	96	3.400	"

TABLE 28. The LC_{50} for various arthropods to fenthion.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	0.015	Sanders, 1969
Stonefly (*Pteronarcys californica*)	24	0.130	Sanders and Cope, 1968
" (*Simocephalus serrulatus*)	48	0.0051	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	0.004	"
Stonefly (*P. californica*)	48	0.030	"
Amphipod (*G. lacustris*)	48	0.070	"
Stonefly (*P. californica*)	48	0.130	"

FORMOTHION

Mammals

The LD_{50} for the rat was 375 to 535 mg/kg to formothion when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to formothion was 0.5 ppm (Alabaster, 1969).

GARDONA

Mammals

The LD_{50} for rats was 4,000 to 5,000 mg/kg to gardona when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for mallards was $>>2,000$ mg/kg; for young pheasants, $\approx 2,000$ mg/kg; and for chukar partridges, $>>2,000$ mg/kg to gardona when the birds were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970).

HEPTACHLOR

Mammals

The LD_{50} for the rat was 90 mg/kg to heptachlor when the mammal was fed the stated dosage orally (Spector, 1955).

Rats fed heptachlor at a rate of 6 mg/kg of body weight for 18 months produced smaller litters and had a higher death rate among their young (Mestitzova, 1967). Prolonged feeding also increased the occurrence of eye cataracts in both parents and offspring.

Birds

Tucker and Crabtree (1970) reported the LD_{50} for young mallards as $\approx 2,080$ mg/kg to heptachlor when the birds were given the stated dosage orally in a capsule. The LC_{50} for mallards was 450 to 700 ppm; for pheasants, 250 to 275 ppm; for bobwhites, 90 to 100 ppm; and for coturnix, 80 to 95 ppm of heptachlor in diets of 2 week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Heptachlor in acetone injected into hen eggs at 400 ppm and 500 ppm killed only 20 and 47 percent of the embryos (Dunachie and Fletcher, 1969).

From 1961 to 1963 a significant increase in the level of residues of heptachlor took place in spring woodcock arrivals in New Brunswick (Wright, 1965). The increase was from an average of 0.5 to 7.2 ppm heptachlor.

In a study of a 2,400 acre cattle farm in Alabama, heptachlor applied at a rate of 2 lb/A for 2 years caused a significant reduction in the songbird population (DeWitt, Stickel and Springer, 1963).

In England wood pigeons were fed under controlled laboratory conditions with heptachlor at dosages of 40, 54, 75, 99, 135, 180, and 245 mg/kg, and the toxicities and residues in flesh and various organs of the birds were measured (Turtle et al., 1963). After this investigation pigeons in the field were examined for the residues they contained. The results of the laboratory and field analyses supported the conclusion that heptachlor was one of the main causes of wood pigeon deaths. These results prompted the government to discontinue the use of heptachlor as a seed dressing.

The effects of heptachlor-contaminated earthworms on woodcocks were investigated in a series of controlled feeding experiments (Stickel, Hayne and Stickel, 1965). Six of the 12 woodcocks receiving heptachlor-treated (2.86 ppm) earthworms died within 35 days; 4 more died by the 53rd day. The last 2 birds were killed for analyses. Earthworms from areas in Louisiana which had been taken from a field treated with heptachlor at 2 lb/A contained more than 3 ppm of heptachlor. Woodcocks were found to eat about 121 g of earthworms per day, or about 77 percent of their body weight.

In bobwhite quail confined to field plots treated with heptachlor, 1½ and 2 lb/A caused heavy mortality, ½ lb/A caused some mortality, and ¼ lb/A caused no mortality. Quail were introduced in pairs and when one died, the other was killed and a new pair introduced. At 2 lb/A one or both members of all pairs introduced within a week of treatment died in less than 18 days. Counting the sacrificed birds as survivors, this represented a 81-percent mortality at 2 lb/A, 50 percent at 1½ lb/A, and 17-percent at ½ lb/A (Kreitzer and Spann, 1968).

Quail populations in Georgia declined significantly soon after the land was treated with heptachlor at a rate of 2 lb/A, and the populations had not yet recovered after a period of 3 years of no further treatment (Rosene, 1965). A decline of cocks and coveys of quail also followed the ¼-lb heptachlor applications (significant for cocks, approaching statistical significance for coveys). A small 4-acre plot within the treated area was searched for dead and dying animals and observations were made on living animals. Forty-seven days after treatment, no live animals were seen or heard on the plot, and a total of 33 dead animals had been found.

A 2-year study carried out to determine the effects heptachlor treatments were having on bird populations in Mississippi disclosed that all treatment rates of heptachlor at 0.25, 0.50, and 2.00 lb/A "decimated arthropod populations, caused bird mortality, and altered bird behavior patterns." None of the dosages, however, eradicated the imported fire ants as planned. There were more bird deaths after the 0.25 application than after the 0.50 and 2.00 treatments. The nesting birds and ground-dwelling insectivorous birds were the most severely affected. Recovery of both insect and bird populations was fairly complete after one year of no insecticide spraying (Ferguson, 1964).

In 1957 the U.S. Department of Agriculture in a cooperative program with the States treated approximately 27 million acres in the Southeast with heptachlor at a rate of 2 lb/A for control of the imported fire ant (Smith and Glasgow, 1963). Investigations of the effects of heptachlor on wildlife were initiated after the second year of treatment in south-central Louisiana. On 4 farms the following animals died within 3 weeks after treatment: 53 mammals, including 12 species; 292 birds, including 29 species; 20 reptiles, including at least 8 species; many species of frogs, many kinds of crayfish; and many fish, including 8 species. Ninety-five percent of the dead animals were analyzed, and all contained some heptachlor. In a study area treated in May 1958 bird-nesting success was only 11.4 percent in 1958 and increased to 45.4 percent in 1959. In a control area the nesting attempts for 1959 were 65 percent.

The responses of different species to a particular pesticide are quite specific, even if they are closely related species. For example, Grolleau and Giban (1966 in Moore, 1967) showed that whereas the closely related gray partridge, red-legged partridge, pheasant, and bantam all react in a similar manner to BHC, they respond quite differently to heptachlor.

Fishes

The LC_{50} of heptachlor tested against various species of fish is found in table 30.

The 24-hour LC_{50} for rainbow trout exposed to heptachlor at temperatures of 1.6°C, 7.2°C, and 12.7°C was 17 ppb, 19 ppb, and 13 ppb, respectively (Macek, Hutchinson and Cope, 1969).

Bluegill growth was reduced in heptachlor-treated (0.05 ppm) ponds, averaging only 7.85 g after 56 days, compared with the controls which grew to 18.5 g in the same time (Cope, 1966).

The toxicity of heptachlor to 2 species of fish, as measured by the 48-hour EC_{50}, was as follows: bluegill at 20 ppm, 24°C, and rainbow trout at 9 ppm, 13°C (Cope, 1966).

Mosquito fish collected in the Mississippi Delta region were resistant to heptachlor, with a 36-hour LC_{50} of 1,300 ppb, whereas the control's was 70 ppb (Boyd and Ferguson, 1964b).

An investigation of the persistence of heptachlor in fish revealed that 50 percent of the chemical was lost in about 1 month (Andrews, Van Valin and Stebbings, 1966).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles exposed to heptachlor was 0.85 ppm (Sanders, 1970).

Arthropods and Annelids

The LC_{50} for various arthropods to heptachlor is found in table 51.

The toxicity of heptachlor to 4 species of arthropods, as measured by the 48-hour EC_{50}, was as follows: waterflea (*Simocephalus serrulatus*) at 47 ppm; waterflea (*Daphnia pulex*) at 42 ppm; mayfly nymph (*Baetis* sp.) at 32 ppm; and stonefly nymph (*Pteronarcys californica* [*sic*]) at 8 ppm (Cope, 1966).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *S. serrulatus* and *D. pulex*, to heptachlor was 47 ppb and 42 ppb, respectively (Sanders and Cope, 1966).

Heptachlor applied to field plots at 5, 10, and 20 lb/A on an Ohio golf course significantly reduced the number of earthworms one year later (Polivka, 1953 in Davey, 1963).

Plants

Heptachlor at 1 ppm in soil significantly suppressed the growth (6.9 g) of corn during 8 weeks' exposure, whereas with heptachlor at 10 and 100 ppm the corn plants weighed significantly more (17.9 g and 17.4 g) than the untreated controls (10.7 g) (Cole et al., 1968). A similar response pattern occurred with beans at the same dosages, the only difference being that at 1 ppm the decrease in growth was less, but not significantly so. At dosages of 1, 10, and 100 ppm in soil heptachlor

TABLE 50. The LC_{50} for various fish to heptachlor.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Rainbow trout	24	0.018	Cope, 1965
Harlequin fish	24	0.09	Alabaster, 1969
Rainbow trout	24	0.25	Mayhew, 1955
Rainbow trout	48	0.009	FWPCA, 1968
Bluegill	96	0.019	Henderson, Pickering and Tarzwell, 1959
Goldfish	96	0.23	"

TABLE 51. The LC_{50} for various arthropods to heptachlor.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcella badia*)	24	0.009	Sanders and Cope, 1968
" (*Pteronarcys californica*)	24	0.008	"
" (*Claassenia sabulosa*)	24	0.009	"
Sand shrimp	24	0.110	Eisler, 1969
Amphipod (*Gammarus lacustris*)	24	0.170	Sanders, 1969
Hermit crab	24	0.460	Eisler, 1969
Grass shrimp	24	>0.5	"
Stonefly (*P. badia*)	48	0.004	FWPCA, 1968
" (*P. californica*)	48	0.006	Sanders and Cope, 1968
Waterflea (*Daphnia pulex*)	48	0.042	FWPCA, 1968
" (*D. pulex*)	48	0.042	Sanders and Cope, 1966
Amphipod (*G. lacustris*)	48	0.100	FWPCA, 1968

caused significant changes in the macro and micro element (N, P, K, Ca, Mg, Mn, Fe, Cu, B, Al, Sr, and Zn) constituents measured in the above ground portions of corn and bean plants (Cole et al., 1968). For example, zinc was significantly higher (89 ppm dry weight) in bean plants treated with 100 ppm of heptachlor compared with the untreated controls (55 ppm); however, nitrogen levels were significantly lower (4.00 percent) in the treated bean plants, compared with the untreated controls (7.25 percent).

Heptachlor applied to soils at 1, 2, or 5 lb/A to a depth of 3 inches was found to be translocated into alfalfa growing on the soil, and the same was observed for cucumbers growing in soil which received 5 to 25 lb/A to a depth of 5 inches (Lichtenstein et al., 1965).

Biological Concentration

Oysters exposed in flowing seawater for 10 days to heptachlor in the water at a dosage of 0.01 ppm concentrated the pesticide in their bodies 17,600 times (176 ppm) (Wilson, 1965).

In pond water containing 0.05 ppm of heptachlor bluegill fish concentrated heptachlor to a level of 15.70 ppm (Cope, 1966).

Peanuts concentrated heptachlor and heptachlor epoxide from soil at a level of 0.16 ppm to 0.67 ppm in peanut meat (Beck et al., 1962).

Seeds with a high oil content, such as soybeans and peanuts, contained nearly 10 times the level of heptachlor and its epoxides than corn seeds with less oil (Bruce, Decker and Wilson, 1966).

Persistence

Heptachlor applied at 20 lb/A persisted in soil for >9 years (Lichtenstein and Polivka, 1959). The heptachlor remaining 14 years after application at a rate of 100 ppm to sandy loam soil was 16 percent (Nash and Woolson, 1967).

ISODRIN

Mammals

The LD_{50} for the rat was 12 to 17 mg/kg to isodrin when the mammal was fed the stated dosage orally (PCOC, 1966).

Persistence

Isodrin applied at 25 ppm to soil persisted (50 percent loss) for >6 years, and isodrin remaining 14 years after application at a rate of 100 ppm to sandy loam soil was 15 percent (Nash and Woolson, 1967).

LEAD ARSENATE

Mammals

The LD_{50} for sheep was 100 mg/kg to lead arsenate when the mammal was fed the stated dosage orally (St. John et al., 1940).

The LD_{50} for lead arsenate administered by oral means to rats and rabbits was 825 and 125 mg/kg, respectively (Metcalf, Flint and Metcalf, 1962).

Coulson, Remington and Lynch (1934) reported that rats fed shrimp which naturally were found to contain 17.70 ppm of arsenic stored only 0.11 mg of arsenic in the 8 months they were fed these shrimp. In a related experiment rats fed lead arsenate at a similar daily dosage stored 5.75 mg. No signs of poisoning were detected in either group.

Birds

Chickens were reported to be able to consume as much as 840 mg of lead arsenate per day for 60 days without suffering noticeable ill effects (Thomas and Shealy, 1932).

The LD_{50} for chickens was 450 mg/kg to lead arsenate when the birds received the stated dosage orally (Metcalf, Flint and Metcalf, 1962).

Plants

The poor condition of alfalfa and barley in a number of unproductive fields in the Yakima Valley where orchards had once stood was attributed to arsenic in the soil (Van de Caveye, Horner and Keaton, 1936). The field contained 4.5 to 12.5 ppm of soluble arsenic. Barley sampled at blossom stage in fields growing in the contaminated soils had from 10.01 to 17.50 ppm As_2O_3 in the tops and 788 to 1,040 ppm in the roots.

Lead arsenate applied at 250, 500, and 1,000 lb/A resulted in residues being detected in vegetables grown in the soil (McLean, Weber and Joffe, 1944). Arsenic trioxide (As_2O_3) detected in the vegetables varied according to vegetable type, with radishes accumulating the largest dosage (0.035 to 0.80 ppm) and snap beans the smallest (none to a trace). On orchard soils onions were found to have high residues of up to 2.26 ppm. The investigators reported that when the arsenic content of the soil is extremely high, plants do not survive.

The effect of lead arsenate added to the soil of orchards annually at 410 lb/A from 1940 to 1953 was measured by growing various crop plants in the contaminated soil for several years after the treatments (MacPhee, Chisholm and MacEachern, 1960). With a mean residue in the soil of about 140 ppm of lead arsenate at time of growth, yields of the crop plants were as follows: beans reduced by 60 percent; turnips increased by 1.8 times; carrots reduced by 30 percent; tomatoes reduced by 36 percent; and peas reduced by 50 percent.

Persistence

Lead arsenate applied at 1,300 lb/A persisted at detectable levels in soil for 15 years (Neiswander, 1951).

LINDANE

Mammals

The LD_{50} for the rat was 125 to 200 mg/kg; for the mouse, 86 mg/kg; for the rabbit, 60 to 200 mg/kg; and for the guinea pig, 100 to 127 mg/kg to lindane (gamma isomer of BHC) when the mammals were fed the stated dosages orally (Spector, 1955).

Birds

Tucker and Crabtree (1970) reported the LD_{50} for young mallards as ≥2,000 mg/kg to lindane when the birds were given the stated dosage orally in a capsule. The LC_{50} for mallards was ≥5,000 ppm; for pheasants, 500 to 600 ppm; for bobwhites, 800 to 1,100 ppm; and for coturnix, 400 to 500 ppm of lindane in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Lindane in acetone injected into hen eggs at 400 ppm and 500 ppm killed 38 and 62 percent of the embryos, respectively (Dunachie and Fletcher, 1969). However, when chicks which had hatched from eggs receiving 100 ppm of lindane were starved for 4 days, all died, whereas in untreated controls only about 50 percent died.

Fishes

The LC_{50} for BHC tested against goldfish was 0.23 ppm, 96 hours exposure (Henderson, Pickering and Tarzwell, 1959), and against rainbow trout, 0.030 ppm, 24 hours exposure (Cope, 1965).

The toxicity of lindane to 2 species of fish, as measured by the 48-hour EC_{50}, was as follows: bluegill at 53 ppb, 24°C, and rainbow trout at 22 ppb, 13°C (Cope, 1966).

As both exposure time and temperature increased, the LC_{50} for bluegills decreased (table 32).

The LC_{50} for various fish to lindane is found in table 33.

About 15 percent of the mosquito fish surviving an exposure to lindane at concentrations producing low mortalities (10 to 40 percent) aborted their young (Boyd, 1964).

In another test the 24-hour LC_{50} for bluegills exposed to lindane at temperatures of 12.7°C, 18.3°C, and 23.8°C was 100 ppb, 100 ppb, and 95 ppb, respectively (Macek, Hutchinson and Cope, 1969).

An investigation of the persistence of lindane in fish revealed that 50 percent of the chemical was lost in <2 days (Gakstatter and Weiss, 1967).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles and chorus frog tadpoles exposed to lindane was 14 ppm and 4.0 ppm, respectively, and the 24-hour LC_{50} for Fowler's toad tadpoles exposed to BHC was 15 ppm (Sanders, 1970).

Arthropods and Annelids

The LC_{50} for various arthropods to lindane is found in table 34.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to lindane was 520 ppb and 460 ppb, respectively (Sanders and Cope, 1966).

TABLE 52. The effect of temperature on the LC_{50} for lindane to small bluegills (about 1 g) (Cope, 1965).

Temperature, °F	LC_{50} (ppb)		
	24 hrs	48 hrs	96 hrs
45	180	88	51
55	100	74	68
65	100	70	58
75	100	53	68
85	94	57	25

TABLE 53. The LC_{50} for various fish to lindane.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Harlequin fish	24	0.075	Alabaster, 1969
Rainbow trout	48	0.018	FWPCA, 1968
Brown trout	96	0.002	Macek and McAllister, 1970
Rainbow trout	96	0.027	"
Largemouth bass	96	0.032	"
Coho salmon	96	0.041	"
Channel catfish	96	0.044	"
Black bullhead	96	0.064	"
Yellow perch	96	0.068	"
Bluegill	96	0.068	"
Redear sunfish	96	0.083	"
Fathead minnow	96	0.087	"
Carp	96	0.090	"
Goldfish	96	0.131	"

TABLE 54. The LC_{50} for various arthropods to lindane.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcys californica*)	24	0.012	Sanders and Cope, 1968
Sand shrimp	24	0.014	Eisler, 1969
Hermit crab	24	0.038	"
Grass shrimp	24	0.062	"
Amphipod (*Gammarus lacustris*)	24	0.120	Sanders, 1969
Stonefly (*P. californica*)	48	0.008	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.088	"
Waterflea (*Daphnia pulex*)	48	0.460	"

The toxicity of lindane to 3 species of arthropods, as measured by the 48-hour EC_{50}, was as follows: waterflea (*S. serrulatus*) at 520 ppb, waterflea (*D. pulex*) at 460 ppb, and stonefly nymph (*P. californicus* [*sic*]) at 9 ppb (Cope, 1966).

Populations of the mite *Tetranychus bimaculatus* increased on beans and potatoes up to 9 times after lindane applications from 0.5 to 15 lb/A (Klostermeyer and Rasmussen, 1953). BHC applied at 3 to 15 lb/A under the same conditions increased mite populations up to 15 times.

The number of dipterans (*Pegomyia hyoscyami*) nearly doubled after a treatment of BHC at a dosage of 28.8 g/m², and this was thought to be due to a reduction in number of predators associated with the dipterans (Lipa, 1958). The number of wireworms, however, apparently decreased from 1.1 per m² to none under this treatment.

Hoy (1955) in New Zealand tested the effects of lindane at 9 and 10 lb/A in soil against earthworms (*Lumbricus rubellus* and *Allolobophora caliginosa*) and observed no significant mortality for 8 weeks.

Treating soil with BHC at a dose of 20.16 g/m² increased the number of earthworms by 2½ times over that of the control (Lipa, 1958).

Lindane, at concentrations of from 0.3 to 0.4 ppb, killed or immobilized 50 percent of the brown and pink shrimp exposed for 48 hours (Butler and Springer, 1963).

Plants

BHC applied as 15 lb/A of gamma isomer in the form of low gamma technical material severely harmed beets, lettuce, and spinach yields (Boswell et al., 1955).

The effect of BHC added to the soil annually at 52 lb/A from 1949 to 1953 was measured by growing various crop plants in the contaminated soil for several years following the treatments (MacPhee, Chisholm and MacEachern, 1960). With a mean residue in the soil of 10.8 ppm of BHC at time of growth, yields of turnips increased by 1.7 times.

After 5 years of cropping only the high dosage of 15 lb/A of BHC significantly reduced the yield of Abruzzi rye grass (Clore et al., 1961).

Phytoplankton exposed for 4 hours to 1 ppm of lindane showed a 28- to 46-percent reduction in productivity (Butler, 1963b).

Biological Concentration

Oysters exposed for 10 days in flowing seawater to lindane at 0.05 ppm in water concentrated the lindane 60 times (3 ppm) (Wilson, 1965).

Persistence

BHC applied at 10 lb/A persisted in soil for ≥11 years (Lichtenstein and Polivka, 1959).

BHC applied at 25 ppm to soil persisted for 9 years, and BHC remaining 14 years after application at a rate of 100 ppm to sandy loam soil was 10 percent (Nash and Woolson, 1967).

MALATHION

Mammals

The LD_{50} for the rat was 480 to 1,500 mg/kg; for the mouse, 885 to 1,120 mg/kg; and for the guinea pig, 570 mg/kg to malathion when the mammals were fed the stated dosages orally (Spector, 1955).

A watershed in Ohio was aerially sprayed with 2 lb/A of malathion, and the effects on many forms of life from microbiota to raccoons were compared with those of an untreated watershed (Peterle and Giles, 1964). Mice and chipmunk populations were reduced in the treated area, but shrews and larger mammals appeared to be unaffected.

Birds

The LD_{50} for young mallards was 1,485 mg/kg to malathion when the birds were fed the stated dosage orally in capsules (Tucker and Crabtree, 1970). The LC_{50} for mallards was >5,000 ppm; for pheasants, 2,500 to 4,500 ppm; for bobwhites, 3,000 to 3,700 ppm; and for coturnix, 2,000 to 2,300 ppm of malathion in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Malathion in acetone injected into hen eggs at 25 ppm, 100 ppm, 200 ppm, 300 ppm, 400 ppm, and 500 ppm killed 15, 18, 38, 29, 58, and 94 percent of the embryos. This toxicant also caused teratogenic effects, especially when combined with thiram in a 3 to 1 ratio (Dunachie and Fletcher, 1969).

When chickens were fed malathion at a dosage of 100 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Birds in the malathion (2 lb/A)-treated watershed area were noticeably quiet for 2 days after the spraying, but otherwise there was no measurable effect (Peterle and Giles, 1964).

Fishes

The toxicity of malathion to 3 species of fishes, as measured by the 48-hour EC_{50}, was as follows: channel catfish at 8,800 ppb, 24°C; bluegill at 86 ppb, 24°C; and rainbow trout at 79 ppb, 13°C (Cope, 1966).

The LC_{50} of malathion tested against various species of fish is found in table 35.

The 24-hour LC_{50} for bluegills exposed to malathion at temperatures of 12.7°C, 18.3°C, and 23.9°C was 220 ppb, 140 ppb, and 110 ppb, respectively (Macek, Hutchinson and Cope, 1969).

The 24-hour LC_{50} for rainbow trout to malathion was 130 ppm at 65°F and the 96-hour LC_{50} was 68 ppm at 55°F (table 36).

TABLE 35. The LC_{50} for various fi[illegible] malathion

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Harlequin fish	24	10	Alabaster, 1969
Brook trout	48	0.0195	FWPCA, 1968
Coho salmon	96	0.101	Macek and McAllister, 1970
Bluegill	96	0.103	"
Redear sunfish	96	0.17	"
Rainbow trout	96	0.170	"
Brown trout	96	0.200	"
Yellow perch	96	0.263	"
Largemouth bass	96	0.285	"
Carp	96	6.59	"
Fathead minnow	96	8.65	"
Channel catfish	96	8.97	"
Goldfish	96	10.7	"
Fathead minnow	96	12.5	Henderson, Pickering and Tarzwell, 1959
Black bullhead	96	12.9	Macek and McAllister, 1970

Little is known about the breakdown products of pesticides and their influence on non-target species. In one of the few investigations Wilson (1966) measured the toxicity of malathion and its metabolites to the fathead minnow. The results shown in table 37 clearly demonstrate that many of the metabolites are quite toxic to the animal.

TABLE 36. The effect of time and temperature on the LC_{50} of malathion to small (about 1 g) rainbow trout (Cope, 1965).

Temperature, °F	LC_{50} (ppb)		
	24 hrs	48 hrs	96 hrs
45	100	76	77
55	85	70	68
65	130	100	110

TABLE 37. Toxicity of malathion and its metabolites to the fathead minnow (Wilson, 1966).

Compound	96-hr LC_{50} (ppm)
Malathion	14
Diethyl succinate	18
Malic acid	25
Mercapto succinic acid	30
Diethyl fumarate	58
Diethyl maleate	41
Dimethyl phosphite	225
Dimethyl phosphate	280

Mount and Stephan (1967) reported that during 7 weeks of exposure to malathion in water at 0.58 ppm fathead minnows had a 20-percent mortality. The authors concluded that the 0.58-ppm concentration is about the maximum at which prolonged survival is possible.

Fish in the streams in the malathion-treated (2 lb/A) watershed area were unaffected by the treatment (Peterle and Giles, 1964).

An investigation of the persistence of malathion in fish revealed that about 50 percent of the chemical was lost in <1 day (Bender, 1969 in Marsh, 1969).

Reptiles and Amphibians

Both reptiles and amphibians in the malathion-treated (2 lb/A) watershed area were unaffected by the treatment (Peterle and Giles, 1964).

The 24-hour LC_{50} for Fowler's toad tadpoles and chorus frog tadpoles exposed to malathion was 1.0 ppm and 0.56 ppm, respectively (Sanders, 1970).

Arthropods and Annelids

The LC_{50} for various arthropods to malathion is found in table 38.

The 48-hour EC_{50} (immobilization value at 60° F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to malathion was 3.5 ppb and 1.8 ppb, respectively (Sanders and Cope, 1966).

TABLE 38. The LC_{50} for various arthropods to malathion.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	96	0.0038	Sanders, 1969
Stonefly (*Pteronarcella badia*)	96	0.010	Sanders and Cope, 1968
" (*Claassenia sabulosa*)	96	0.013	"
" (*Pteronarcys californica*)	96	0.035	"
Hermit crab	96	0.116	Eisler, 1969
Grass shrimp	96	0.161	"
Sand shrimp	96	0.246	"
Waterflea (*Daphnia pulex*)	48	0.0018	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.0018	"
Waterflea (*D. pulex*)	48	0.002	Cope, 1966
Stonefly (*Simocephalus serrulatus*)	48	0.003	"
" (*P. badia*)	48	0.008	FWPCA, 1968
Mayfly (*Baetis* sp.)	48	0.006	Cope, 1966
Stonefly (*P. californicus* [sic])	48	0.020	"
Red crawfish	48	>50.0	Muncy and Oliver, 1963

Martin and Wiggins (1959) found that the manure worm immersed for 2 hours in malathion tolerated 0.1 ppm, but was killed with the 1 ppm dosage.

Malathion as a drift contaminant caused an outbreak in the cottony-cushion insect scale through the destruction of its vedalia beetle predator (Bartlett and Lagace, 1960). The authors remarked that malathion was then recommended at a higher dosage for control of the very problem it had caused.

Crayfish in streams in the malathion-treated (2 lb/A) watershed were unaffected by the treatment (Peterle and Giles, 1964). Other arthropod numbers, however, decreased greatly, but recovered soon after the treatment.

Large numbers of honeybees were killed after the application of malathion (8 fluid oz/A) for grasshopper control in alfalfa (Levin et al., 1968). Malathion "residues were detected in alfalfa (12-20 ppm), in pollen (0.43-11.1 ppm), and in dead bees (<0.01-0.37 ppm) for as long as 8 days after the application."

Plants

Malathion at 0.1 ppm appeared to be converted to malaoxon and other metabolites by the algae and also altered the composition of the mixed algal community to which it was added. There was no persistent inhibitory effect on algal growth (Christie, 1969).

Persistence

Malathion applied to soil persisted for 2 days (Lange and Schulz, 1958).

Malathion applied at 5 lb/A (about 3.2 ppm) persisted for 8 days (about 0.1 ppm remaining) in a silt-loam soil (Lichtenstein and Schulz, 1964).

MECARBAM

Mammals

The LD_{50} for rats was 36 mg/kg to mecarbam when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

Mecarbam in acetone injected into hen eggs at 50 ppm, 100 ppm, and 200 ppm killed 26, 52, and 87 percent of the embryos, respectively (Dunachie and Fletcher, 1969). This toxicant also caused teratogenic effects at 200 ppm.

MENAZON

Mammals

The LD_{50} for female rats was 1,950 mg/kg to menazon when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

Menazon in acetone injected into hen eggs at 10 ppm, 50 ppm, 100 ppm, and 200 ppm killed 5, 32, 60, and 70 percent of the embryos, respectively (Dunachie and Fletcher, 1969). This toxicant also caused teratogenic effects at 500 ppm and above.

When chickens were fed menazon at a dosage of 400 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The 24-hour LC_{50} for harlequin fish to menazon was about 210 ppm (Alabaster, 1969).

METACIDE

Mammals

Metacide was reported to be somewhat less toxic to warm-blooded animals than parathion (FCH, 1970).

Annelids

Metacide at 1 and 2.5 percent reduced earthworms (*Caloglyphus anomalus*) in 7 days by 97 and 100 percent, respectively (Hyche, 1956). At 0.06 percent all earthworms survived.

METHOMYL

Mammals

The LD$_{50}$ for rats was 17 to 24 mg/kg (FCH, 1970) and for mule deer, 11.0 to 22.0 mg/kg (Tucker and Crabtree, 1970) to methomyl when the mammals were fed the stated dosages orally.

Birds

The LD$_{50}$ for young mallards was 15.9 mg/kg to methomyl when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

METHOXYCHLOR

Mammals

The LD$_{50}$ for the rat was 5,000 to 6,000 mg/kg and for the mouse, 300 mg/kg to methoxychlor when the mammals were fed the stated dosages orally (Spector, 1955).

Birds

The LD$_{50}$ for young mallards was >2,000 mg/kg to methoxychlor when the birds were fed the stated dosage orally in capsules (Tucker and Crabtree, 1970). The LC$_{50}$ for mallards was >5,000 ppm; for pheasants, >5,000 ppm; for bobwhites, >5,000 ppm; and for coturnix, >5,000 ppm of methoxychlor in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Methoxychlor in acetone injected into hen eggs at up to 500 ppm caused little or no mortality to the embryos (Dunachie and Fletcher, 1969).

Methoxychlor was found to be much less toxic to robins than DDT in both laboratory and field tests (Hunt and Sacho, 1969). The authors reported that "it was impossible to produce methoxychlor poisoning consistently with dosages as high as 3,750 mg/kg." Elms treated with DDT at dosages of greater than 10 lb/A caused spring mortalities in the robin population of over 85 percent. When methoxychlor was substituted at similar rates for DDT, the mortality was reduced to 24 percent.

Fishes

The LC$_{50}$ for various fish to methoxychlor is found in table 39.

The 24-hour LC$_{50}$ for rainbow trout exposed to methoxychlor at temperatures of 1.6°C, 7.2°C, and 12.7°C was 55 ppb, 45 ppb, and 74 ppb, respectively (Macek, Hutchinson and Cope, 1969); and the 24-hour LC$_{50}$ for bluegills exposed at temperatures of 12.7°C, 18.3°C, and 23.8°C was 58 ppb, 67 ppb, and 88 ppb, respectively.

About 15 percent of the mosquito fish surviving an exposure to methoxychlor with low mortalities (10 to 40 percent) were observed to abort their young (Boyd, 1964).

Amphibians

The 24-hour LC$_{50}$ for Fowler's toad tadpoles and chorus frog tadpoles exposed to methoxychlor was 0.76 ppm and 0.44 ppm, respectively (Sanders, 1970).

Arthropods

The LC$_{50}$ for various arthropods to methoxychlor is found in table 40.

The 48-hour EC$_{50}$ (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to methoxychlor was 5 ppb and 0.78 ppb, respectively (Sanders and Cope, 1966).

When methoxychlor was used as a blackfly (*Simulium venustum*) larvicide, it produced residues in the treated water bodies lower than those that occur with DDT (Burdick et al., 1968). The effect of methoxychlor on stream arthropods appeared to be about the same as that of DDT.

TABLE 39. The LC$_{50}$ for various fish to methoxychlor.

Fish Species	Exposure Period (hr)	LC$_{50}$ (ppm)	Source
Rainbow trout	24	0.052	Mayhew, 1955
Rainbow trout	48	0.0072	FWPCA, 1968
Goldfish	96	0.056	Henderson, Pickering and Tarzwell, 1959
Guppies	96	0.120	"

TABLE 40. The LC_{50} for various arthropods to methoxychlor.

Arthropod Species	Exposure (hours)	LC_{50} (ppm)	Source
Amphipod (*Gammarus fasciatus*)	24	0.0047	Sanders, 1969
Sand shrimp	96	0.004	Eisler, 1969
Hermit crab	96	0.007	"
Grass shrimp	96	0.012	"
Stonefly (*Pteronarcys californica*)	96	0.080	Sanders and Cope, 1968
" (*Pteronarcys* sp.)	96	0.030	Cope, 1965
Waterflea (*Daphnia pulex*)	48	0.00078	Sanders and Cope, 1966
" (*D. pulex*)	48	0.0008	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.0013	"
Stonefly (*P. californica*)	48	0.0047	Sanders, 1969
" (*P. californica*)	96	0.008	FWPCA, 1968

Plants

The exposure of phytoplankton communities for 4 hours to 1 ppm of methoxychlor reduced their productivity 80.6 percent (Butler, 1963a).

Biological Concentration

When oysters were exposed in flowing seawater for 10 days to methoxychlor at 0.05 ppm in water, they concentrated the toxicant 5,780 times (289 ppm) (Wilson, 1965).

The exposure of brook trout to methoxychlor at 0.005 ppm in water resulted in their accumulating an average of 1.750 ppm during 7 days (Burdick et al., 1968). This was much less than occurred with DDT at the same dosage and time (DDT in fish averaged 2.946 ppm). When placed in fresh water, the fish lost 47.3 percent of the accumulated methoxychlor within one week.

MEVINPHOS

Mammals

The LD_{50} for rats was 6.8 mg/kg to mevinphos when the mammals were fed the stated dosage orally (USDI, 1970).

Birds

The LD_{50} for young mallards was 4.6 mg/kg; for young pheasants, 1.4 mg/kg; and for sharp-tailed grouse, 0.75 to 1.50 mg/kg to mevinphos when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The LC_{50} for rainbow trout and bluegills to mevinphos for a 24-hour exposure was 34 ppb and 41 ppb, respectively (Cope, 1965).

The 48-hour LC_{50} for rainbow trout exposed to mevinphos was 17 ppb (FWPCA, 1968).

The 24-hour LC_{50} for harlequin fish to mevinphos was 18 ppm (Alabaster, 1969).

Arthropods

The LC_{50} for various arthropods to mevinphos is found in table 41.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to mevinphos was 0.43 ppb and 0.16 ppb, respectively (Sanders and Cope, 1966).

TABLE 41. The LC_{50} for various arthropods to mevinphos

Arthropod Species	Exposure Period (hr)	LC_{50} (ppm)	Source
Sand shrimp	24	0.013	Eisler, 1969
Hermit crab	24	0.040	"
Stonefly (*Pteronarcys* sp.)	24	0.058	Cope, 1965
" (*P. californica*)	24	0.030	Sanders and Cope, 1968
Grass shrimp	24	0.101	Eisler, 1969
Amphipod (*Gammarus lacustris*)	24	0.600	Sanders, 1969
Waterflea (*Daphnia pulex*)	48	0.00016	FWPCA, 1968
Stonefly (*P. californica*)	48	0.009	"
Amphipod (*G. lacustris*)	96	0.010	"

MILBEX

Mammals

The LD_{50} for the mouse was 800 mg/kg to milbex when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to milbex was 4.1 ppm (Alabaster, 1969).

MIREX

Mammals

The LD_{50} for the rat was 300 to 600 mg/kg to mirex when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Mirex fed to mice at 5 ppm in their diet resulted in reduced litter size and number of offspring produced per day (Ware and Good, 1967).

Birds

The LD_{50} for young mallards was >2,400 mg/kg to mirex when the birds were given the stated dosage orally in capsules (Tucker and Crabtree, 1970). The LC_{50} for pheasants was 1,400 to 1,600 ppm, but for coturnix, >10,000 ppm of mirex in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Hens fed mirex at 300 and 600 ppm in their diets lost weight, and the survival of their chicks was reduced (Naber and Ware, 1965).

Fishes

Juvenile striped mullet exposed to a variety of chlorinated hydrocarbon insecticides at concentrations ranging from 0.4 to 7 ppb for 48 hours had a 50-percent mortality with most insecticides (Butler and Springer, 1965). The exceptions were mirex, BHC, chlordecone, lindane, and methoxychlor; concentrations of these materials had to be 10 to 100 times greater to kill the same 50 percent of this fish species.

Bluegill growth was reduced significantly when exposed for up to 168 days to mirex at 5 ppm (Van Valin, Andrews and Eller, 1968). No effect was observed at dosages of 1 and 3 ppm of mirex.

Arthropods

The LC_{50} for mirex for 48 hours exposure against red crawfish was greater than 0.1 ppm (Muncy and Oliver, 1963).

Plants

The exposure of phytoplankton for 4 hours to 1 ppm of mirex reduced their productivity 20 to 40 percent (Butler, 1963b).

Biological Concentration

In pond water containing 1 ppm of mirex, bluegill fish concentrated the mirex to a level of 6.8 ppm (Cope, 1966).

NABAM

Birds

When chickens were fed nabam at a dosage of 400 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

MONOCROTOPHOS

Mammals

The LD_{50} for rats was about 21 mg/kg (PCH, 1970); for domestic goats, 20 to 50 mg/kg; and for mule deer, 25 to 50 mg/kg (Tucker and Crabtree, 1970) to monocrotophos when the animals were fed the stated dosages orally in capsules.

Birds

The LD_{50} for young mallards was 4.8 mg/kg; for young pheasants, 2.8 mg/kg; for young chukar partridges, 6.5 mg/kg; for young coturnix, 3.7 mg/kg; for bobwhite, 0.9 mg/kg; for pigeons (*Columba livia*), 2.8 mg/kg; for house sparrows, 1.6 mg/kg; for house finches, 8 to 24 mg/kg; for Canada geese, 1.6 mg/kg; and for golden eagle, <0.75 mg/kg to monocrotophos when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to monocrotophos was 7 ppm (FWPCA, 1968).

MORPHOTHION

Birds

Morphothion in acetone injected into hen eggs at 10 ppm, 50 ppm, and 100 ppm killed 16, 21, and 57 percent of the embryos, respectively (Dunachie and Fletcher, 1969). This toxicant also caused some teratogenic effects at 50 ppm and above.

NALED

Mammals

The LD_{50} for the rat was 430 mg/kg (PCH, 1970), and for mule deer, ~200 mg/kg (Tucker and Crabtree, 1970) to naled when the mammals were given the stated dosages orally in a capsule.

Birds

The LD_{50} for mallards was 52.2 mg/kg; for sharp-tailed grouse, 64.9 mg/kg; and for Canada geese, 36.9 mg/kg to naled when the birds were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970). The LC_{50} for mallards was >5,000 ppm; for pheasants, 2,400 to 2,700 ppm; for bobwhites, 2,400 to 3,100 ppm; and for coturnix, 1,200 to 1,400 ppm of naled in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Fishes

The LC_{50} for bluegills to naled for 24-hour exposure was 0.220 ppm (Cope, 1965).

The 24-hour LC_{50} for rainbow trout exposed to naled at temperatures of 1.6°C, 7.2°C, and 12.7°C was 1,000 ppb, 620 ppb, and 240 ppb, respectively (Macek, Hutchinson and Cope, 1969).

The 48-hour LC_{50} for brook trout exposed to naled was 78 ppb (FWPCA, 1968).

Amphibians

The 24-hour LC_{50} for chorus frog tadpoles exposed to naled was 2.2 ppm (Sanders, 1970).

Arthropods

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to naled was 1.1 ppb and 0.35 ppb, respectively (Sanders and Cope, 1966).

The LC_{50} for various arthropods to naled is found in table 65.

TABLE 63. The LC_{50} for various arthropods to naled

Arthropod Species	Exposure time (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcys californica*)	96	0.007	Sanders and Cope, 1968
Amphipod (*Gammarus lacustris*)	96	0.380	Sanders, 1969
Waterflea (*Daphnia pulex*)	48	0.0003	FWPCA, 1968
Stonefly (*P. californica*)	48	0.016	"
Amphipod (*G. lacustris*)	48	0.180	"
Red crawfish	48	3.0	Muncy and Oliver, 1963

NICOTINE

Mammals

The LD_{50} for rats was 50 to 60 mg/kg to nicotine when the mammals were fed the stated dosages orally (FCH, 1970). Hayne (1949) reported that nicotine sulfate (40 percent) at one-half teaspoon in a quart of water applied as a spray to beans and cabbage effectively repelled cottontail rabbits.

Birds

The LD_{50} for young mallards was 507 mg/kg; for young pheasants, 1,000 to 2,000 mg/kg; for young coturnix, 330 mg/kg; and for pigeons (*Columba livia*), >2,000 mg/kg to nicotine sulfate when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Nicotine in acetone injected into hen eggs at 10 ppm, 15 ppm, 25 ppm, 50 ppm, and 100 ppm killed 0, 20, 100, 100, and 100 percent of the embryos, respectively (Dunachie and Fletcher, 1969). This toxicant also caused teratogenic effects at 25 ppm.

N-METHYL CARBAMATE

Fishes

The 24-hour LC_{50} for harlequin fish to N-methyl carbamate was 0.61 ppm (Alabaster, 1969).

OVEX

Mammals

The LD_{50} for rats was 2,050 mg/kg to ovex when the mammals were fed the stated dosage orally (FCH, 1970).

Fishes

The 48-hour LC_{50} for bluegill exposed to ovex was 700 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for stoneflies (*Pteronarcys californica*) exposed to ovex was 1,500 ppb (FWPCA, 1968).

OXYDEMETON METHYL

Mammals

The LD_{50} for rats was 70 mg/kg to oxydemeton methyl when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for young mallards was 53.9 mg/kg; for young pheasants, 42.4 mg/kg; for young chukar partridges, 113 mg/kg; for young coturnix, 84.1 mg/kg; for pigeons (*Columba livia*), 14.9 mg/kg; and for house sparrows, 70.8 mg/kg to oxydemeton methyl when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Arthropods

The estimated 24-hour LC_{50} for stonefly nymphs (*Pteronarcys californica*) to oxydemeton methyl was 800 ppb (Sanders and Cope, 1968).

The 96-hour LC_{50} for an amphipod (*Gammarus lacustris*) exposed to oxydemeton methyl was 190 ppb (Sanders, 1969).

OXYTHIOQUINOX

Mammals

The LD$_{50}$ for rats was about 3,000 mg/kg to oxythioquinox when the mammals were fed the stated dosage orally (FCH, 1970).

Fishes

The 48-hour LC$_{50}$ for bluegill exposed to oxythioquinox was 96 ppm (FWPCA, 1968).

Arthropods

The 48-hour LC$_{50}$ for stoneflies (*Pteronarcys californica*) exposed to oxythioquinox was 40 ppm (FWPCA, 1968).

Frequent applications of oxythioquinox to orchards at recommended dosages were found to destroy predaceous mite populations; however, the insecticide was generally harmless to the beneficial parasites *Marmoniella* and *Aphelinus* (Besemer, 1964).

PARAOXON

Mammals

The LD$_{50}$ for the rat was 3.5 mg/kg to paraoxon when the mammal was fed the stated dosage orally (PCOC, 1966).

Persistence

Persistence of paraoxon in water at 20°C was 330 days (Muhlmann and Schrader, 1957).

PARATHION

Mammals

The LD$_{50}$ for the rat was 4 to 30 mg/kg; for the mouse, 25 mg/kg; for the guinea pig, 32 mg/kg (Spector, 1955); for domestic goats, 28 to 56 mg/kg; and for mule deer, 22 to 44 mg/kg (Tucker and Crabtree, 1970) to parathion (ethyl) when the mammals were fed the stated dosages orally.

Populations of the white-footed mouse in New Jersey woods adjacent to treated crop fields were exposed to parathion at 0.01 to 0.06 lb/A and DDT at 0.12 to 0.21 lb/A (Jackson, 1952). Because the level of contamination of the adjacent woods was low and the ingestion of insecticides was quite small, the mouse population was not measurably affected.

Birds

The LD$_{50}$ for young mallards was 1.9 to 2.1 mg/kg; for young pheasants, 12.4 mg/kg; for young chukar partridges, 24.0 mg/kg; for young coturnix, 6.0 mg/kg; for pigeons (*Columba livia*), 2.5 mg/kg; for sharp-tailed grouse, 4.0 to 10.0 mg/kg; for house sparrows, 3.4 mg/kg; for young gray partridges, 16.0 mg/kg; and for fulvous tree ducks, 0.12 to 0.25 mg/kg to parathion when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970). The LC$_{50}$ for mallards for 250 to 275 ppm; for pheasants, 350 to 380 ppm; for bobwhites, 180 to 200 ppm; and for coturnix, 40 to 50 ppm of parathion in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Tucker and Crabtree (1970) also reported that the LD$_{50}$ for young mallards was 10.0 mg/kg and for young pheasants, 8.2 mg/kg to methyl parathion when the birds were given the stated dosages orally in a capsule. The LC$_{50}$ for mallards was 600 to 750 ppm; for pheasants, 100 to 120 ppm; for bobwhites, 90 to 100 ppm; and for coturnix, 45 to 55 ppm of methyl parathion in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Parathion in acetone injected into hen eggs at 10 ppm, 50 ppm, 100 ppm, 200 ppm, 300 ppm, 400 ppm, and 500 ppm killed 0, 11, 35, 48, 50, 41, and 81 percent of the embryos, respectively (Dunachie and Fletcher, 1966). This toxicant also caused some teratogenic effects at 300 ppm.

When chickens were fed methyl parathion at a dosage of 64 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

In the Union of South Africa a large-scale spray program was carried out on a citrus estate using parathion applied at 7.5 lb/A for control of citrus scale insects, especially *Aonidiella aurantii* (Buttiker, 1961). In the treated orchard nearly 200 birds were found dead, including the following (plus 20 miscellaneous species): Kurrichane thrush, yellow-eye, Jardine's babbler, blue waxbill, Melba finch, green white-eye, and speckled coly.

Mallard ducks appeared to be unaffected when duck ponds were treated with parathion at a dosage of 0.85 lb/A (Mulla, 1966).

In another study ethyl and methyl parathion were applied at rates of ½ and 8 lb/A (USDI, 1966). There was no effect on pheasants from the ethyl parathion at ½ lb, but the 8-lb rate killed about 10 percent. Methyl parathion, however, killed about 9 percent at the ½-lb rate and about 25 percent at the 8-lb rate.

Fishes

The 96-hour LC_{50} for parathion tested against fathead minnow was 1.4 to 2.7 ppm (Henderson, Pickering and Tarzwell, 1959). The 96-hour LC_{50} for minnows to parathion was 1,780 ppb (Priester, 1965). No parathion was detected in minnows which had been fed parathion-treated *Daphnia*. Other LC_{50} values for various fish to parathion are found in table 43.

In tests, the 96-hour LC_{100} of parathion was 1 ppm for carp, 0.5 ppm for tilapia, and 0.125 ppm for mullet (Lahav and Sarig, 1969c); the highest nonlethal dosage was 0.5 ppm for carp, 0.25 ppm for tilapia, and 0.1 ppm for mullet.

An investigation of the persistence of parathion in fish revealed that 50 percent of the chemical was lost in <1 week (Miller, Zuckerman and Charig, 1966).

When a cranberry bog was treated with parathion at a concentration of 0.12 ppm, 80 percent of the fish (*Fundulus heteroclitus*) were killed (Miller, Zuckerman and Charig, 1966). The parathion in the ponds decreased to an insignificant level within about 144 hours after treatment.

All mosquito fish were killed when exposed to water in duck ponds treated with parathion at a dosage of 0.85 lb/A (Mulla, 1966).

Molluscs

Freshwater mussels survived a concentration of 0.12 ppm parathion in a cranberry bog (Miller, Zuckerman and Charig, 1966).

Amphibians

The 24-hour LC_{50} for chorus frog tadpoles exposed to parathion was 1.0 ppm (Sanders, 1970).

Frogs survived well in duck ponds treated with parathion at a dosage of 0.85 lb/A (Mulla, 1966).

Arthropods and Annelids

The LC_{50} of parathion tested against various species of arthropods is found in table 44.

TABLE 43. The LC_{50} for various fish to parathion

Formulation	Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Ethyl	Bluegill	48	0.047	FWPCA, 1968
Ethyl	Rainbow trout	48	2	Sanders, 1969
Methyl	Bluegill	48	8	FWPCA, 1968
Methyl	Rainbow trout	96	2.75	Macek and McAllister, 1970
Methyl	Yellow perch	96	3.06	"
Methyl	Brown trout	96	4.74	"
Methyl	Redear sunfish	96	5.17	"
Methyl	Largemouth bass	96	5.22	"
Methyl	Coho salmon	96	5.3	"
Methyl	Channel catfish	96	5.71	"
Methyl	Bluegill	96	5.72	"
Methyl	Black bullhead	96	6.64	"
Methyl	Carp	96	7.13	"
Methyl	Fathead minnow	96	8.9	"
Methyl	Goldfish	96	9.0	"

TABLE 44. The LC_{50} for various arthropods to parathion.

Formulation	Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Ethyl	Stonefly (*Pteronarcella badia*)	24	0.008	Sanders and Cope, 1968
Ethyl	" (*Claassenia sabulosa*)	24	0.0088	"
Methyl	Sand shrimp	24	0.011	Eisler, 1969
Ethyl	Amphipod (*Gammarus lacustris*)	24	0.012	Sanders, 1969
Methyl	Grass shrimp	24	0.015	Eisler, 1969
Methyl	Hermit crab	24	0.023	"
Ethyl	Stonefly (*Pteronarcys californica*)	24	0.028	Sanders and Cope, 1968
Ethyl	Waterflea (*Daphnia pulex*)	48	0.0004	FWPCA, 1968
Ethyl	" (*Daphnia* sp.)	48	0.00078	Priester, 1965
Methyl	" (*D. magna*)	48	0.0048	FWPCA, 1968
Ethyl	Amphipod (*G. lacustris*)	48	0.006	"
Ethyl	Stonefly (*Pteronarcys* sp.)	48	0.011	Cope, 1965
Ethyl	" (*P. californica*)	48	0.011	FWPCA, 1968
Methyl	Red crawfish	48	0.04	Muncy and Oliver, 1963

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to parathion was 0.37 ppb and 0.60 ppb, respectively (Sanders and Cope, 1966).

No parathion could be detected in *Daphnia* after 7 days of exposure to 0.5 ppb (Priester, 1965).

When cole plants were treated with parathion (0.20 lb/A) and endrin (0.18 lb/A), only 5 percent of the total number of predaceous and parasitic insect species survived, compared with the number in the untreated control. However, 92 percent of the plant-feeding species survived the insecticide treatment (Pimentel, 1961). The predaceous and parasitic species were probably lost in the treated plots because of the severe reduction in the numbers of individual prey, resulting in many plant-feeding species with few predaceous and parasitic species.

Hyche (1956) reported that parathion applied to soil as a 1-percent, 0.06-percent, and 0.003-percent emulsion killed 100, 17, and 0 percent, respectively, of the earthworm *Caloglyphus* [illegible] population.

The dispersion and biological effects of parathion moving from a treated peach orchard into a 2.7-acre pond were investigated in South Carolina (Nicholson et al., 1962). During the active spray season parathion in the water rose to a maximum of 1.22 ppb, whereas during the winter the concentration was as low as 0.01 ppb. Prior to the spray season (March) parathion was found in the mud at a level of 1.90 ppm. Investigators felt this was a carry-over from the spray operation of the previous season. Fish, zooplankton, adult aquatic insects, and Oligochaeta populations appeared to be unaffected by the residues of parathion in the water and mud. There was, however, a significant reduction in immature aquatic insect numbers associated with parathion usage.

Biological Concentration

Fish (*Fundulus heteroclitus*) concentrated parathion 80 times that of the ambient water level when exposed to 0.12 ppm of parathion; and mussels concentrated parathion 50 times the level in the water (Miller, Zuckerman and Charig, 1966). Labelled S^{35} was employed to track the movement of the parathion from the water into the organisms.

Persistence

The persistence of parathion in water at 20°C was 600 days, and of methyl parathion, 175 days (Muhlmann and Schrader, 1957).

Parathion applied to soil persisted for 5 years (MacPhee, Chisholm and MacEachern, 1960); and parathion and methyl parathion applied at 5 lb/A (about 5.2 ppm) persisted for 90 days and 30 days, respectively (about 0.1 ppm remaining), in a silt-loam soil (Lichtenstein and Schulz, 1965).

PARIS GREEN

Mammals

The LD$_{50}$ for the rat was 22 mg/kg to Paris green when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LC$_{50}$ for mallards was >5,000 ppm; for pheasants, 1,000 to 1,100 ppm; for bobwhites, 500 to 600 ppm; and for coturnix, 1,200 to 1,400 ppm of Paris green in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

Amphibians

The median lethal dose of Paris green to frogs by subcutaneous injection was 10 mg/kg (Spector, 1955).

PERTHANE

Mammals

The LD$_{50}$ for the rat was 8,170 mg/kg to perthane when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Birds

The LC$_{50}$ for mallards was ≥5,000 ppm; for pheasants, ≥5,000 ppm; and for coturnix, ≥5,000 ppm of perthane in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

Fishes

The LC$_{50}$ for rainbow trout to perthane for a 24-hour exposure was 9 ppb (Cope, 1965).

The 48-hour LC$_{50}$ for rainbow trout exposed to perthane was 7 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC$_{50}$ for waterfleas (*Daphnia magna*) exposed to perthane was 9.4 ppb (FWPCA, 1968).

PHORATE

Mammals

The LD$_{50}$ for the rat was 3.7 mg/kg to phorate when the mammal was fed the stated dosage orally (USDI, 1970).

Birds

Tucker and Crabtree (1970) reported the LD$_{50}$ for young mallards as 0.62 mg/kg; for young pheasants, 7.1 mg/kg; and for young chukar partridges, 12.8 mg/kg to phorate when the birds were fed the stated dosages orally in capsules. The LC$_{50}$ for mallards was 225 to 275 ppm; for pheasants, 400 to 450 ppm; and for bobwhites, 370 to 400 ppm of phorate in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

When chickens were fed phorate at a dosage of 62 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

Fishes

The 48-hour LC$_{50}$ for bluegill exposed to phorate was 5.5 ppb (FWPCA, 1968). The 24-hour LC$_{50}$ for harlequin fish to phorate was <1 ppm (Alabaster, 1969).

Amphibians

The LD$_{50}$ for bullfrogs was 55.2 mg/kg to phorate when the frogs were fed the stated dosage orally (Tucker and Crabtree, 1970).

Arthropods

The 48-hour LC$_{50}$ for amphipods (*Gammarus lacustris*) exposed to phorate was 70 ppb (FWPCA, 1968).

The 24-hour LC_{50} for an amphipod (*G. lacustris*) exposed to phorate was 24 ppb (Sanders, 1969).

Persistence

Phorate applied to soil persisted for >28 days (Laygo and Schulz, 1963).

PHOSALONE

Fishes

The 24-hour LC_{50} for harlequin fish to phosalone was 3.4 ppm (Alabaster, 1969).

PHOSPHAMIDON

Mammals

The LD_{50} for the rat was 27 mg/kg to phosphamidon when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Birds

The LD_{50} for young mallards was 3.0 mg/kg; for young chukar partridges, 0.7 mg/kg; for pigeons (*Columba livia*), 2 to 3 mg/kg; for mourning doves, 2 to 4 mg/kg; for white-winged doves, 2.5 mg/kg to phosphamidon when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was 700 to 800 ppm; for pheasants, 70 to 80 ppm; for bobwhites, 20 to 30 ppm; and for coturnix, 100 to 110 ppm of phosphamidon in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

Phosphamidon in acetone injected into hen eggs at 15 ppm, 25 ppm, 50 ppm, and 100 ppm killed 71, 42, 88, and 100 percent of the embryos, respectively (Dunachie and Fletcher, 1969). This toxicant also caused teratogenic effects at 15 ppm.

In a field trial with phosphamidon applied at a rate of 1 lb/A to 5,000 acres in Montana against spruce budworm, blue grouse were killed, and other bird activity dropped by about one-quarter of the pre-spray level (Finley, 1965). Bird activity in the untreated area, however, increased during the same period.

Phosphamidon, suggested as a substitute for control of gypsy moth, was found to be extremely toxic to quail (USDI, 1967). Quail were found unable to survive on diets containing 1 ppm of phosphamidon.

In Switzerland many bird deaths were reported after the treatment of 2,022 acres of larch forest with phosphamidon at a rate of 0.8 lb/A (Schneider, 1966).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to phosphamidon was 8,000 ppb (FWPCA, 1968).

Phosphamidon applied at a rate of 1 lb/A for control of the spruce budworm in forested areas had no apparent harmful effects on young Atlantic salmon and brook trout (Kerswill and Edwards, 1967).

Arthropods

The LC_{50} for various arthropods to phosphamidon is found in table 45.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to phosphamidon was 12.0 ppb and 8.8 ppb, respectively (Sanders and Cope, 1966).

TABLE 45. The LC_{50} for various arthropods to phosphamidon

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	0.0094	Sanders, 1969
Stonefly (*Pteronarcys californica*)	24	1.4	Sanders and Cope, 1968
Amphipod (*G. lacustris*)	48	0.0038	FWPCA, 1968
Waterflea (*Daphnia magna*)	48	0.004	"
Stonefly (*P. californica*)	48	0.460	"
Red crawfish	48	6.0	Muncy and Oliver, 1963

One year after the treatment of northwestern Ontario forests with phosphamidon at 4 oz/A and fenitrothion at 6 oz/A the long-term effects were evaluated on predaceous carabid beetles and lycosid spiders (Freitag and Poulter, 1970). The populations of these predators were clearly suppressed in the treated area. The investigators stated that the results did "not imply a 1 year persistence of the insecticides, but rather a persistent disturbance of the ecosystem."

PIPERONYL BUTOXIDE

Mammals

The LD$_{50}$ for rats was 11,500 mg/kg to piperonyl butoxide when the mammals were fed the stated dosage orally (FCH, 1970).

Amphibians

The 24-hour LC$_{50}$ for chorus frog tadpoles exposed to piperonyl butoxide was 1.8 ppm (Sanders, 1970).

PROPOXUR

Mammals

The LD$_{50}$ for rats was 100 mg/kg (Neumeyer, Gibbons and Trask, 1969); for domestic goats, >800 mg/kg; and for the mule deer, 100 to 350 mg/kg to propoxur when the animals were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970).

Birds

The LD$_{50}$ for young mallards was 11.9 mg/kg; for young pheasants, 20 mg/kg; for young chukar partridges, 25.9 mg/kg; for coturnix, 28.3 mg/kg; for California quail, 30 mg/kg; for pigeons (*Columba livia*), 60.4 mg/kg; for mourning doves, 4.2 mg/kg; for house sparrows, 12.8 mg/kg; for house finches, 3.6 mg/kg; for Oregon juncos, 4.8 mg/kg; for Canada geese, 6.0 mg/kg; and for lesser sandhill cranes, 40 to 60 mg/kg to propoxur when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The 48-hour LC$_{50}$ for fathead minnow exposed to propoxur was 25 ppb (FWPCA, 1968).

Amphibians

The LD$_{50}$ for bullfrogs was 595 mg/kg to propoxur when the animals were fed the stated dosage orally (Tucker and Crabtree, 1970).

Arthropods

The 48-hour LC$_{50}$ for stoneflies (*Pteronarcys californica*) and amphipods (*Gammarus lacustris*) exposed to propoxur was 110 ppb and 25 ppb, respectively (FWPCA, 1968). The 24-hour LC$_{50}$ for an amphipod (*G. lacustris*) exposed to propoxur was 66 ppb (Sanders, 1969).

PYRETHRINS

Mammals

The LD$_{50}$ for the rat was 820 to 1,870 mg/kg and for the guinea pig, 150 mg/kg to pyrethrins when the mammals were fed the stated dosages orally (Spector, 1955).

Birds

The LD$_{50}$ for young mallards was >10,000 mg/kg to pyrethrins when the birds were fed the stated dosage orally in capsules (Tucker and Crabtree, 1970).

Fishes

The 48-hour LC$_{50}$ for rainbow trout exposed to pyrethrins was 54 ppb (FWPCA, 1968).

Arthropods

The LC$_{50}$ for various arthropods to pyrethrins is found in table 46.

TABLE 46. The LC_{50} for various arthropods to pyrethrins.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcys californica*)	24	0.010	Sanders and Cope, 1968
Amphipods (*Gammarus fasciatus*)	24	0.028	Sanders, 1969
" (*G. lacustris*)	48	0.018	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	0.025	"
Stonefly (*P. californica*)	48	0.004	"

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to pyrethrins was 42 ppb and 25 ppb, respectively (Sanders and Cope, 1966).

The toxicity of pyrethrins to 3 species of invertebrates as measured by the 48-hour EC_{50} was as follows: waterflea (*S. serrulatus*) at 42 ppb, waterflea (*D. pulex*) at 25 ppb, and stonefly nymph (*P. californicus* [*sic*]) at 6 ppb (Cope, 1966).

RONNEL

Mammals

The LD_{50} for rats was 1,740 mg/kg to ronnel when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

When chickens were fed ronnel at a dosage of 1,000 mg/kg, the chickens developed leg weakness (Gaines, 1969). Mode of action was unknown.

ROTENONE

Mammals

The LD_{50} for the rat was 132 mg/kg to rotenone when the mammal was fed the stated dosage orally (Spector, 1955).

Birds

The LD_{50} for young mallards was >2,000 mg/kg and for young pheasants, >1,414 mg/kg to rotenone when the birds were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970).

Rotenone in acetone injected into hen eggs at 1 ppm, 5 ppm, and 10 ppm killed 30, 80, and 100 percent of the embryos, respectively (Dunachie and Fletcher, 1969).

Fishes

The LC_{50} for coho salmon embryos to rotenone for a 24-hour exposure was 0.15 ppm (Garrison, 1968); embryos could survive a concentration of 0.075 ppm of rotenone for 24 hours; however, 100-day-old fry could survive only at a dosage of 0.00375 ppm.

The 48-hour LC_{50} for bluegill exposed to rotenone was 22 ppb (FWPCA, 1968).

Arthropods, Annelids, and Other Invertebrates

The LC_{50} for various arthropods to rotenone is found in table 47.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to rotenone was 190 ppb and 100 ppb, respectively (Sanders and Cope, 1966).

In New Zealand earthworms were destroyed when 4-percent rotenone was applied at a rate of about 6 lb/A, and the soil remained toxic for at least 6 days (Harris, 1949).

The minimum lethal dosages (ppm) of rotenone producing a kill exceeding 25 percent are listed for the following fish-food organisms: *Daphnia*, 0.1; *Eucypris*, 0.1; *Hyallella*, 0.2; *Palaemonetes*, 1.0; *Amphiagrion*, 2.5; *Pachydiplax* and *Tramea*, 3.5; *Culex*, *Aedes*, and *Anopheles*, 2.0; *Chironomus*, 0.1; *Physa*, 4.5; and *Helisoma*, 5.5 (Hischkale, 1952).

TABLE 47. The LC_{50} for various arthropods to rotenone.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcys* sp.)	24	3.9	Cope, 1965
" (*P. californica*)	24	2.9	Sanders and Cope, 1968
Amphipod (*Gammarus lacustris*)	24	5	Sanders, 1969
Waterflea (*Daphnia pulex*)	48	0.010	FWPCA, 1968
Amphipod (*G. lacustris*)	48	0.350	"
Stonefly (*P. californica*)	48	0.900	"

When rotenone was applied to cole crops, cabbage aphid and peach aphid outbreaks followed (Pimentel, 1961). Although the parasites were also abundant in the rotenone-treated area compared with the untreated control, the ratio of parasites to aphids was significantly lower in the rotenone area.

Rotenone applied at 1.5 ppm to lakes appeared to have an inhibitory effect on 3 groups of plankton (Entomostraca, Rotatoria, and Protozoa) which are important fish foods (Hoffman and Olive, 1961).

SCHRADAN

Mammals

The LD_{50} for rats was 8 to 25 mg/kg to schradan when the mammals were fed the stated dosages orally (FCH, 1973).

Birds

The LD_{50} for young mallards was 36.5 mg/kg to schradan when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Schradan in acetone injected into hen eggs at 10 ppm, 15 ppm, 50 ppm, and 100 ppm killed 15, 60, 100, and 95 percent of the embryos, respectively (Dunachie and Fletcher, 1969). This toxicant also caused teratogenic effects at 25 ppm and above.

TDE

Mammals

The LD_{50} for the rat was 3,360 to 3,400 mg/kg and for the mouse, 2,280 mg/kg to TDE when the mammals were fed the stated dosages orally (Spector, 1955).

Birds

The LC_{50} for mallards was 4,800 to 5,000 ppm; for pheasants, 500 to 600 ppm; for bobwhites, 2,200 to 2,400 ppm; and for coturnix, 3,000 to 3,500 ppm of TDE in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

TDE in acetone injected into hen eggs at up to 500 ppm killed only 10 percent of the embryos (Dunachie and Fletcher, 1969). However, when chicks which had hatched from eggs receiving 100 ppm of TDE were starved for 4 days, all died, whereas untreated controls handled in a similar manner resulted in only about a 50-percent mortality.

TDE in concentrations of 10 and 40 ppm in the feed did not cause demonstrable changes in eggshell thickness of mallards, but did impair reproductive success of mallard ducks by nearly 50 percent (significant $P<0.05$) (Heath, Spann and Kreitzer, 1969).

TDE and DDE were fed separately to cowbirds (Stickel, Stickel and Coon, 1970). The residues of these chemicals in the brains of birds killed by the toxicants were distinctly higher than in brains of cowbirds sacrificed after similar exposure. TDE residues in the brain at death were estimated to be 65 ppm (wet weight) or higher in 95 percent of the cases, whereas DDE residues in the brain at death were estimated to be 314 ppm or higher in 95 percent of the cases.

Fishes

The LC_{50} for bluegills to TDE for a 24-hour exposure was 56 ppb (Cope, 1965).

The 48-hour LC_{50} for rainbow trout exposed to TDE was 9 ppb (FWPCA, 1968).

In another study about 5 percent of the mosquito fish surviving after exposure to TDE at concentrations above the threshold toxicity aborted their young (Boyd, 1964).

TDE was applied to Clear Lake in California for gnat control at a rate which would produce a concentration of 0.014 ppm for the first application and 0.02 ppm for the last 2 applications. TDE residues in the flesh of the 1958 year class of largemouth bass decreased from 23.5 ppm in 1958 to 7 ppm in 1963 (Linn and Stanley, 1969). After 18 months residue levels of TDE in various organisms in the lake were as follows: 10 ppm in plankton, 903 ppm in fat of plankton-eating fish, 2,690 ppm in fat of carnivorous fish, and 2,134 ppm in fat of fish-eating birds (Hunt and Bischoff, 1960). The residues in the carnivorous fish and birds represent a 100,000-fold increase over the levels of TDE found in the lake water immediately after treatment.

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles and chorus frog tadpoles exposed to TDE was 0.70 ppm and 0.61 ppm, respectively (Sanders, 1970).

Arthropods

The LC_{50} for various arthropods to TDE is found in table 48.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to TDE was 4.5 ppb and 3.2 ppb, respectively (Sanders and Cope, 1966).

TABLE 48. The LC_{50} for various arthropods to TDE

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	0.0056	Sanders, 1969
Stonefly (*Pteronarcys californica*)	24	3	Sanders and Cope, 1968
Amphipod (*G. lacustris*)	48	0.0018	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	0.0032	"
Stonefly (*P. californica*)	48	1.1	"

TEPP

Mammals

The LD_{50} for the rat was 1.2 to 2.0 mg/kg to TEPP when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for young mallards was 3.6 mg/kg; for young pheasants, 4.2 mg/kg; and for young chukar partridges, 10.1 mg/kg to TEPP when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The 48-hour LC_{50} for fathead minnows exposed to TEPP was 380 ppb (FWPCA, 1968).

Amphibians

The LD_{50} for bullfrogs was 80.1 mg/kg to TEPP when the frogs were fed the stated dosage orally in capsules (Tucker and Crabtree, 1970).

Arthropods

The 48-hour LC_{50} for amphipods (*Gammarus lacustris*) exposed to TEPP was 52 ppb (FWPCA, 1968).

The 24-hour LC_{50} for an amphipod (*G. lacustris*) exposed to TEPP was 74 ppb (Sanders, 1969).

TERPENE POLYCHLORINATES

Mammals

The LD_{50} for the rat was 220 mg/kg to terpene polychlorinates when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Birds

The LC_{50} for mallards was 470 to 500 ppm; for pheasants, 500 to 600 ppm; for bobwhites, 600 to 800 ppm; and for coturnix, 500 to 600 ppm of terpene polychlorinates in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

Fishes

The LC_{50} for bluegills to terpene polychlorinates for a 24-hour exposure was 15 ppb (Cope, 1965).

The 48-hour LC_{50} for rainbow trout exposed to terpene polychlorinates was 2.5 ppb (FWPCA, 1968).

A natural population of mosquito fish in ditches adjacent to cotton fields was resistant to terpene polychlorinates (500 times) (Boyd and Ferguson, 1964a). Interestingly enough, the fish had not been exposed to terpene polychlorinates. They had, however, been exposed to toxaphene, a related insecticide.

Arthropods

The LC_{50} for nymphs of the stonefly (*Pteronarcys* sp.) to terpene polychlorinates for a 24-hour exposure was 40 ppb (Cope, 1965).

The 48-hour LC_{50} for stoneflies (*P. californica*) exposed to terpene polychlorinates was 7 ppb (FWPCA, 1968).

The estimated 24-hour LC_{50} for stonefly nymphs (*P. californica*) to terpene polychlorinates was 40 ppb (Sanders and Cope, 1968).

TETRADIFON

Mammals

The LD_{50} for the rat was 14,700 mg/kg to tetradifon when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; and for coturnix, >5,000 ppm of tetradifon in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970a).

Fishes

The 48-hour LC_{50} for bluegill exposed to tetradifon was 1,100 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for amphipods (*Gammarus lacustris*) exposed to tetradifon was 140 ppb (FWPCA, 1968).

The 24-hour LC_{50} for an amphipod (*G. lacustris*) exposed to tetradifon was 370 ppb (Sanders, 1969).

THANITE

Arthropods

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) exposed to Thanite was 460 ppb (FWPCA, 1968).

TOXAPHENE

Mammals

The LD_{50} for the rat was 60 mg/kg; for the mouse, 112 mg/kg; for the dog, 15 mg/kg; for the guinea pig, 69 mg/kg (Spector, 1955); for domestic goats, >160 mg/kg; and for mule deer, 139 to 240 mg/kg (Tucker and Crabtree, 1970) to toxaphene when the mammals were given the stated dosages orally in a capsule.

Birds

Tucker and Crabtree (1970) reported the LD_{50} for young mallards as 70.7 mg/kg; for young pheasants, 40.0 mg/kg; for young bobwhite quail, 66.4 mg/kg; for sharp-tailed grouse, 10 to 20 mg/kg; for fulvous tree ducks, 99.0 mg/kg; and for lesser sandhill cranes, 100 to 316 mg/kg to toxaphene when the birds were fed the stated dosages orally in capsules. The LC_{50} for pheasants was 500 to 550 ppm, and for coturnix, 608 to 650 ppm of toxaphene in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

The LC_{50} for bobwhite quail chicks was 834 ppm and for mallard ducklings, 538 ppm to toxaphene when the birds were fed the stated dosages in their food for 5 days and then fed clean food for 3 days (Heath and Stickel, 1965).

Toxaphene in acetone injected into hen eggs at up to 500 ppm caused no mortality to the embryos (Dunachie and Fletcher, 1969).

When a marsh in North Dakota was treated with toxaphene at 2 lb/A (105 ppm in water), sora, coot, and black tern produced no young; only the red-wing blackbird produced any young (Hanson, 1952).

Pheasants were maintained on a diet containing various levels of toxaphene for periods of time ranging up to 96 days. Of the 55 birds, all survived the exposure period, even at the highest dosage of 300 ppm; however, pheasants at this dosage did lose weight (Genelly and Rudd, 1956).

Toxaphene reportedly limited the reproduction of bobwhite quail and pheasant by at least 25 percent when they were fed a diet containing 50 ppm (bobwhite) and 25 ppm (pheasant) of toxaphene (USDI, 1960).

For 3 months groups of 5 young white pelicans were fed sardines injected with either 10 ppm of toxaphene, 50 ppm of toxaphene, 50 ppm of DDT, or a combination of 10 ppm of toxaphene and 150 ppm of DDT. These dosages were somewhat comparable to those present in fish eaten by wild birds. Death occurred in the pelicans fed toxaphene at the 50 ppm level 4 to 8 weeks after the experiment had started. The pelicans were much more susceptible to toxaphene than DDT (Flickinger and Keith, 1964). Incidentally, both endo- and ectoparasites were nearly completely eliminated from these birds after exposure to the toxaphene-DDT combination (Keith, 1966b).

Unusually high mortalities were observed in fish-eating birds at the Tule Lake and Lower Klamath Refuges in 1960, 1961, and 1962 (Keith, 1966b). The mortalities were reportedly due to applications of large quantities of toxaphene in the agricultural lands immediately surrounding the refuge for several years starting in 1956. Water from the surrounding farmlands drains into the marshes. Toxaphene was found in fish from the marshes at levels of about 8 ppm (Keith, Mohn and Ise, 1965). In another part of California toxaphene was reported causing high mortalities in fish-eating white pelicans (Keith, 1963).

Fishes

The LC_{50} of toxaphene tested against various species of fishes is found in table 40.

The toxicity of toxaphene to 2 species of fish, as measured by the 48-hour EC_{50}, was as follows: rainbow trout at 4 ppb, 13°C, and bluegill at 4 ppb, 24°C (Cope, 1966).

The 24-hour LC_{50} for bluegills exposed to toxaphene at temperatures of 12.7°C, 18.3°C, and 23.8°C was 9.7 ppb, 8.5 ppb, and 6.6 ppb, respectively (Macek, Hutchinson and Cope, 1969).

Toxaphene has been reported to have a high toxicity to fish. The minimum toxic dosage of tox-

TABLE 40. The LC_{50} for various fish to toxaphene

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Rainbow trout	24	0.03	Mayhew, 1955
Rainbow trout	48	0.0028	FWPCA, 1968
Largemouth bass	96	0.002	Macek and McAllister, 1970
Brown trout	96	0.003	"
Bluegill	96	0.0035	Henderson, Pickering and Tarzwell, 1959
Carp	96	0.004	Macek and McAllister, 1970
Black bullhead	96	0.005	"
Goldfish	96	0.0056	Henderson, Pickering and Tarzwell, 1959
Coho salmon	96	0.008	Macek and McAllister, 1970
Rainbow trout	96	0.011	"
Yellow perch	96	0.012	"
Channel catfish	96	0.013	"
Redear sunfish	96	0.013	"
Goldfish	96	0.014	"
Fathead minnow	96	0.014	"
Bluegill	96	0.018	"

aphene for small fish was 0.005 ppm and for larger fish, about 0.007 ppm. When water turbidity was high, concentrations as high as 0.02 ppm were needed to clear the lake of fish (Stringer and McMynn, 1960).

As both temperature and exposure time increased, the LC_{50} for rainbow trout to toxaphene declined (table 60).

Mosquito fish surviving an exposure to toxaphene above the threshold toxicity of the compound aborted (about 5 percent) their young (Boyd, 1964). For the bluntnose minnow, the 96-hour median tolerance limit to toxaphene increased from 0.020 ppm in hard water to 0.058 ppm in soft water at 50°F (Hooper and Grzenda, 1957).

A natural population of mosquito fish in ditches adjacent to cotton fields was found to be resistant to toxaphene (40 times) (Boyd and Ferguson, 1964a).

TABLE 60. The effects of time and temperature on the LC_{50} of toxaphene to small (about 1 g) rainbow trout (Cope, 1965)

Temperature, °F	LC_{50} (ppb) 24 hr	48 hr	96 hr
45	10.0	8.4	5.4
55	7.8	4.4	2.7
65	5.0	2.8	1.8

Three species of fish were collected in the field at Twin Bayou, Mississippi, where the populations had been exposed to heavy concentrations of several insecticides used in the adjoining cotton acreages (Ferguson et al., 1965b). The toxicity of toxaphene in these fish compared with that in a control population, as measured by 36-hour LC_{50}, were: golden shiner, control 20 ppb versus Twin Bayou 1,000 ppb; bluegills, control 23 ppb versus Twin Bayou 1,000 ppb; and green sunfish, control 38 ppb versus Twin Bayou 1,500 ppb. In another investigation the toxicity of toxaphene in resistant mosquito fish and black bullheads collected from streams in Mississippi compared with that in an unexposed control population, as measured by 36-hour LC_{50}, was: mosquito fish, control 20 ppb versus resistant (Sidon, Miss.) 480 ppb; and black bullhead, control 5.75 ppb versus resistant (Wayside, Miss.) 80 ppb (Ferguson et al., 1965a).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles and chorus frog tadpoles exposed to toxaphene was 0.60 ppm and 1.7 ppm, respectively (Sanders, 1970).

Molluscs

After 4 weeks of exposure to 0.1 ppm of toxaphene, 50 percent of the oyster population died (USDI, 1960). Only 1 ppb inhibited the development of clam eggs by 50 percent and also reduced the growth of mature oysters after 7 days of exposure by 64 percent (USDI, 1960). Molluscs in lakes, however, were apparently unaffected by a dosage of 0.1 ppm toxaphene (Hooper and Grzenda, 1957).

The snail population in a marsh treated with toxaphene at 2 lb/A (100 ppm in water) declined slowly to zero in about 10 days (Hanson, 1952). The snails did not start to reinvade the treated area until a month had passed.

Arthropods, Annelids, and Other Invertebrates

The LC_{50} of toxaphene tested against various species of arthropods is found in table 61.

The 48-hour EC_{50} (immobilization value at 60° F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to toxaphene was 19 ppb and 15 ppb, respectively (Sanders and Cope, 1966).

Certain aquatic Oligochaetes in lakes were apparently unaffected by a toxaphene treatment of 0.1 ppm (Hooper and Grzenda, 1957).

Brown shrimp tolerated toxaphene at a dosage of 40 to 50 ppb, whereas white shrimp had a toleration limit of 75 to 90 ppb (USDI, 1960).

Toxaphene at 0.1 ppm appeared to have an inhibitory effect on 3 groups of plankton (Entomostraca, Rotatoria, and Protozoa) which are important fish foods (Hoffman and Olive, 1961).

Toxaphene (10 to 60 μg/beetle) was found to prevent oviposition in coccinellid beetles (*Coleomegilla maculata*) (Atallah and Newsom, 1966).

The bottom fauna in a lake with a 10 ppb level of toxaphene declined in number of individuals, but returned to normal density within 14 days (Hooper, 1960).

TABLE 31. The LC_{50} for various arthropods to toxaphene.

Arthropod Species	Exposure (hours)	LC_{50} (ppm)	Source
Stonefly (*Claassenia sabulosa*)	24	0.006	Sanders and Cope, 1968
" (*Pteronarcella badia*)	24	0.0093	"
" (*Pteronarcys californica*)	24	0.018	"
Amphipod (*Gammarus lacustris*)	24	0.180	Sanders, 1969
Stonefly (*P. californica* (old))	48	0.007	Cope, 1966
" (*P. californica*)	48	0.007	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	0.015	Cope, 1966
" (*D. pulex*)	48	0.015	FWPCA, 1968
" (*Simocephalus serrulatus*)	48	0.019	Cope, 1966
Mayfly (*Baetis* sp.)	48	0.007	"
Amphipod (*G. lacustris*)	48	0.070	FWPCA, 1968

Plants

Exposing phytoplankton communities for 4 hours to 1 ppm of toxaphene reduced the productivity of these communities 91 percent (Butler, 1963a).

In laboratory studies planktonic animals and algae, periphyton, and insect nymphs were exposed to toxaphene in both single doses of 0.03 ppm and chronic doses of 0.01 and 0.02 ppm (Schoettger and Olive, 1961). Single sublethal doses of toxaphene were insufficient to produce toxic accumulations in these organisms.

Biological Concentration

Toxaphene at chronic doses of 0.01 to 0.02 ppm became concentrated in *Daphnia* and periphyton (Schoettger and Olive, 1961). The chronic doses accumulated by *Daphnia* and periphyton were at levels toxic to fish.

Two mountain lakes were treated with toxaphene to eradicate the fish and subsequently investigated to follow the movement and fate of toxaphene in the lakes (Terriere et al., 1966). The concentration in the shallow eutrophic lake, initially treated with about 88 ppb of toxaphene in 1961, decreased to 0.83 ppb in 1962, to 0.41 ppb in 1963, and to 0.02 ppb in 1964. The concentration in the deep oligotrophic lake, initially treated with about 40 ppb in 1961, declined to 2.10 ppb in 1962, to 1.20 ppb in 1963, and to 0.84 ppb in 1964. Both plants and animals absorbed toxaphene and apparently played an important role in eliminating it from the lakes. Plants in the deep lake with water containing about 2 ppb levels of toxaphene concentrated it to levels as high as 17 ppm, while invertebrates concentrated toxaphene to maximum levels of 6 ppm (Terriere et al., 1966). In the shallow lake the concentration factor was about 500 times for aquatic plants, 1,500 times for aquatic invertebrates, and 15,000 times for rainbow trout. In the deeper lake, trout could not be restocked in the lake for 6 years, although the concentration 6 years after treatment had decreased to 0.04 ppb.

In a similar investigation by Kallman, Cope and Navarre (1962), a shallow lake received a treatment of 0.05 ppm of toxaphene. Within 1 month the concentration of toxaphene declined to 0.001 ppm and held at about this level for an additional 200 days. Mortalities of 100 percent were common after 24 hours of exposure to 0.01 ppm. Substantiating findings in the previous study (Terriere et al., 1966), aquatic vegetation concentrated toxaphene to high levels (400 times that found in the water).

Toxaphene was applied to Big Bear Lake, in California, at a calculated rate of 0.2 ppm (Hunt and Keith, 1963). After the treatment, the results of an analysis for toxaphene in sample organisms removed from the lake were as follows: a high of 73 ppm in plankton, 200 ppm in goldfish (the target fish of this control program), and 1,700 ppm in the fat of a pelican.

Oysters exposed to toxaphene at 0.05 ppm for 10 days concentrated the toxicant 3,000 times (148 ppm) (Wilson, 1965).

Persistence

Toxaphene applied at 140 ppm to soil persisted for 14 years (Westlake and San Antonio, 1960).

Toxaphene applied at 10 ppm to soil persisted (50 percent loss) for about 11 years, and toxaphene remaining 14 years after application at a rate of 100 ppm to sandy loam soil was 45 percent (Nash and Woolson, 1967).

TRICHLORFON

Mammals

The LD_{50} for the rat was 450 mg/kg to trichlorfon when the mammal was fed the stated dosage orally (Metcalf, Flint and Metcalf, 1962).

Birds

The LC_{50} for bobwhite was 700 to 800 ppm, and for coturnix, 1,000 to 2,000 ppm of trichlorfon in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Trichlorfon in acetone injected into hen eggs at 100 ppm killed 77 percent of the embryos (Dunachie and Fletcher, 1969).

Fishes

The LC_{50} for various fish to trichlorfon is found in table 52.

In a study 1-inch rainbow trout larvae were exposed for 16 hours to 10, 20, 50 and 100 ppm of trichlorfon and for 40 hours to 5 ppm trichlorfon (Matton and Lattam, 1969). These treatments produced marked inhibition of acetylcholinesterase. The treatments also caused the trout to become hyperactive, no longer to avoid light, and to have a weaker touch stimulus.

Arthropods

The LC_{50} of trichlorfon tested against various species of arthropods is found in table 53.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to trichlorfon was 0.70 ppb and 0.18 ppb, respectively (Sanders and Cope, 1966).

TABLE 52. The LC_{50} for various fish to trichlorfon

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Striped bass	24	10.8	Wellborn, 1969
Rainbow trout	96	3.2	Sanders and Cope, 1968
Fathead minnow	96	180.0	Henderson, Pickering and Tarzwell, 1959

Persistence

The persistence at detectable levels of trichlorfon in water at 20°C was 526 days (Muhlmann and Schrader, 1957).

VAMIDOTHION

Fishes

The 24-hour LC_{50} for harlequin fish to vamidothion was 500 ppm (Alabaster, 1969).

TABLE 53. The LC_{50} for various arthropods to trichlorfon

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcella badia*)	24	0.070	Sanders and Cope, 1968
Amphipod (*Gammarus lacustris*)	24	0.092	Sanders, 1969
Stonefly (*Claassenia sabulosa*)	24	0.110	Sanders and Cope, 1968
" (*Pteronarcys californica*)	24	0.320	"
Waterflea (*Daphnia pulex*)	48	0.00018	Sanders and Cope, 1966
" (*D. magna*)	48	0.0001	FWPCA, 1968
Stonefly (*P. badia*)	96	0.023	"
Amphipod (*G. lacustris*)	96	0.040	"
Stonefly (*P. californica*)	96	0.180	Sanders and Cope, 1968

ZECTRAN

Mammals

The LD_{50} for rats was 19 mg/kg; for dogs, 15 to 30 mg/kg (FCH, 1970); for domestic goats, 15 to 30 mg/kg; and for mule deer, 30 to 50 mg/kg (Tucker and Crabtree, 1970) to zectran when the mammals were given the stated dosages orally in a capsule.

Birds

The LD_{50} for young mallards was 3.0 mg/kg; for pheasants, 4.5 mg/kg; for young chukar partridges, 5.9 mg/kg; for young coturnix, 3.2 mg/kg; for sharp-tailed grouse, 10.0 mg/kg; for pigeons (*Columba livia*), 6.5 mg/kg; for young mourning doves, 2.8 mg/kg; for house sparrows, 50.4 mg/kg; for house finches, 4.8 mg/kg; for Canada geese, 3.6 mg/kg; and for lesser sandhill cranes, 1.0 to 4.5 mg/kg to zectran when the birds were fed the stated dosages orally in capsules (Tucker and Crabtree, 1970). The LC_{50} for mallards was 590 to 880 ppm and for pheasants, 880 to 900 ppm of zectran in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a). Note the disparity between these results and those of Tucker and Crabtree.

Zectran and its metabolites were also found to cause a syndrome in mallard ducks much like diabetes mellitus (USDI, 1966).

Fishes

The LC_{50} for various fish to zectran is found in table 54.

Amphibians

The LD_{50} for bullfrogs was 283 to 900 mg/kg to zectran when the frogs were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Arthropods

The LD_{50} for various arthropods to zectran is found in table 55.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to zectran was 13 ppb and 10 ppb, respectively (Sanders and Cope, 1966).

TABLE 54. The LC_{50} for various fish to zectran.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Rainbow trout	48	8	FWPCA, 1968
Coho salmon	96	1.74	Macek and McAllister, 1970
Yellow perch	96	2.48	"
Brown trout	96	8.1	"
Rainbow trout	96	10.2	"
Bluegill	96	11.2	"
Channel catfish	96	11.4	"
Carp	96	13.4	"
Largemouth bass	96	14.7	"
Black bullhead	96	16.7	"
Redear sunfish	96	16.7	"
Fathead minnow	96	17.0	"
Goldfish	96	19.14	"

TABLE 55. The LC_{50} for various arthropods to zectran.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcys californica*)	24	0.032	Sanders and Cope, 1968
Amphipod (*Gammarus lacustris*)	24	0.086	Sanders, 1969
Waterflea (*Daphnia pulex*)	48	0.010	FWPCA, 1968
Stonefly (*P. californica*)	48	0.018	"
Amphipod (*G. lacustris*)	48	0.076	"

ZINC CHLORIDE

Fishes

The 24-hour LC_{50} for harlequin fish to zinc chloride was 0.17 ppm (Alabaster, 1969).

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PART III
Herbicides

ACROLEIN

Mammals

The LD_{50} for the rat was 46 mg/kg to acrolein when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 24-hour LC_{50} for rainbow trout to acrolein was 0.14 ppm (Alabaster, 1969).

AMETRYNE

Mammals

The LD_{50} for rats was 1,110 mg/kg and for mice, 965 mg/kg to ametryne when the mammals were fed the stated dosages orally (WSA, 1967).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to ametryne was 3,400 ppb (FWPCA, 1968).

Spot exposed to 1.0 ppm of ametryne for 48 hours showed no noticeable effects (Butler, 1963).

Molluscs

Eastern oysters exposed to 1.0 ppm of ametryne for 96 hours exhibited no restriction of shell growth (Butler, 1963).

Arthropods

When brown shrimp were exposed to 1.0 ppm of ametryne for 48 hours, a 10-percent mortality or paralysis resulted (Butler, 1963).

AMIBEN

Mammals

The LD_{50} for the rat was 5,620 mg/kg to amiben when the mammal was fed the stated dosage orally (PCOC, 1966).

Persistence

Amiben applied at 2 to 5 lb/A persisted in soil for >6 weeks (Anderson, 1965), and when applied to soil (concentration not stated) persisted for about 3 months (Kearney, Nash and Isensee, 1969).

α-AMINO-2,6-DICHLORO-BENZALDOXINE

Fishes

The 24-hour LC_{50} for harlequin fish to α-amino-2,6-dichloro-benzaldoxime and its hydrochloride formulation was 350 ppm and 240 ppm, respectively (Alabaster, 1969).

AMITROLE

Mammals

The LD_{50} for rats was 5,000 mg/kg to amitrole when the mammals were fed the stated dosage orally (USDA, 1967 in House et al., 1967).

Birds

The LD_{50} for mallard ducks was >2,000 mg/kg to amitrole when the ducks were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; and for coturnix, >5,000 ppm of amitrole in diets of 2-week-old birds when fed treated food for 5 days followed by clean food for 3 days (Heath et al., 1970).

Several species of birds survived when fed diets containing as much as 5,000 ppm of various herbicides; however, amitrole was found to depress reproduction in mallard ducks fed dosages at least 25 percent below those which would produce mortality (USDI, 1962).

Fishes

Hiltibran (1967) reported that bluegill, green sunfish, lake chub sucker, and smallmouth bass fry survived a concentration of 50 ppm of amitrole for 8 days or the termination of the experiment.

The estimated 48-hour LC_{50} to salmon was 3,250 ppm for amitrole (Bohmont, 1967).

Arthropods and Nematodes

The median immobilization concentration for amitrole to *Daphnia magna* was 23 ppm (Crosby and Tucker, 1966).

Courtney, Peabody and Austenson (1962) reported that amitrole applied at a rate of 8 lb/A to bentgrass reduced the number of nematodes in the bentgrass by 40 percent.

Microorganisms

The microbial degradation of dalapon was inhibited in the presence of amitrole, but amitrole degradation was not hindered by the presence of dalapon (Kaufman, 1966). The phytotoxic residues of both dalapon and amitrole persisted in the soil longer applied in combination than applied separately. Dalapon, especially, disappeared more slowly when amitrole also had been applied to the soil.

Persistence

Amitrole applied at 20 ppm persisted at detectable levels in soil for 7 weeks (Hurschel and Freed, 1959).

Amitrole applied at a rate of 2 to 10 lb/A to moist loam was found to persist for 3 to 5 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

Amitrole applied at a rate of 1.0 ppm persisted in the water for more than 200 days with significant quantities of the herbicide being detected in the hydrosoil (Grzenda, Nicholson and Cox, 1966).

AMMONIUM THIOCYANATE

Mammals

Ammonium thiocyanate used as both a fungicide and herbicide was observed to be effective in repelling porcupines (Welch, 1954 in Springer, 1957). Livestock reportedly avoided eating treated vegetation (FCH, 1970).

Fishes

A concentration of 200 ppm ammonium thiocyanate proved lethal to fish in Russian studies (Demyanenko, 1941 in Springer, 1957).

Persistence

Ammonium thiocyanate applied at 500 ppm persisted at detectable levels in soil for 6 to 8 weeks (Newton and Paul, 1965).

AMS

Mammals

The LD_{50} for the rat was 3,900 mg/kg to AMS when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to AMS was 1,250 ppm (Alabaster, 1969).

Persistence

AMS in soil persisted for 1 to 3 months (Hurd-Karrer, 1946).

ASULAM

Mammals

The LD_{50} for the rat was 5,000 mg/kg to asulam when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to asulam (potassium salt) was 5,200 ppm (Alabaster, 1969).

ATRAZINE

Mammals

The LD_{50} for the rat was 3,080 mg/kg and for the mouse, 1,750 mg/kg to atrazine when the mammals were fed the stated dosages orally (WSA, 1967).

Birds

The LD_{50} for mallards was >2,000 mg/kg to atrazine when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; and for bobwhites, 700 to 800 ppm of atrazine in diets of 2-week-old birds when fed treated food for 5 days followed by untreated food for 3 days (Heath et al., 1970).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to atrazine was 12,600 ppb (FWPCA, 1968). The 24-hour LC_{50} for harlequin fish to atrazine was 0.55 ppm (Alabaster, 1969).

Spot exposed to 1.0 ppm of atrazine for 48 hours exhibited no deleterious effect (Butler, 1963).

Hiltibran (1967) reported that bluegill and green sunfish fry survived a concentration of 10 ppm of atrazine (wettable powder) for 8 days or the termination of the experiment.

Mollusca

After the application of atrazine at dosages of 0.5 to 2.0 ppm to pond enclosures, clams were reduced to about ½ their original number, whereas the snail population increased nearly 4 times (Walker, 1962).

The exposure of eastern oysters to 1.0 ppm of atrazine for 96 hours had no noticeable effect on shell growth (Butler, 1963).

Arthropods, Annelids, and Other Invertebrates

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) exposed to atrazine was 3,600 ppb (FWPCA, 1968).

The following species of bottom organisms were reduced by at least 50 percent after the application of atrazine in dosages ranging from 0.5 to 2 ppm: waterbugs, mayfly nymphs, horsefly larvae, common midges (Tendipedidae), mosquitoes, phantom midges, biting midges, caddice fly larvae, aquatic worms (Oligochaeta), and leeches (Walker, 1962). In contrast, damselfly nymphs and water beetles actually doubled their numbers after the application.

Brown shrimp, exposed to 1.0 ppm of atrazine for 48 hours, experienced 20 percent mortality or paralysis (Butler, 1963).

In plots treated with normal dosages (2 to 4 lb/A in WSA, 1967) of atrazine, wireworms, earthworms, and springtails declined in numbers, whereas the numbers of mites and millipedes remained about the same (Fox, 1964). The author pointed out that it was possible that the changes in soil fauna were due to changes in the composition of the vegetation and not due to the direct effects of atrazine.

Edwards (1964) reported that atrazine at normally recommended dosages did not cause a significant reduction in the numbers of soil animals.

Plants

Synergism between atrazine at ½ lb/A and each of the following herbicides was detected: dextron at ½ oz/A, lasso at 4 oz/A, diphenamid at 2 oz/A, nitralin at 1 oz/A, 2,4-D at 1 oz/A, trifluralin at 2 oz/A, nitralin and dextron at 1 + ½ oz/A, and diphenamid and trifluralin at 2 + 2 oz/A (Lynch, Sweet and Dickerson, 1970). These combinations were found to be from 2 to 10 times more effective against the bean test plant than atrazine and oil alone.

Microorganisms

Some acceleration of nitrification was observed in soil treated with atrazine, but the total production of nitrates did not increase (Balicka and Sobieszczanski, 1969a in Balicka, 1969).

During 4 years of applying atrazine at a rate of 4.4 lb/A, no change in the number of microorganisms in the soil was found, regardless of the medium used for microorganism determination (Balicka and Sobieszczanski, 1969b in Balicka, 1969).

Atrazine at normal dosages (2 to 4 lb/A in WSA, 1967) in soil caused an increase in the number of *Azotobacter* (Balicka, 1969).

Persistence

Atrazine applied at 2 lb/A persisted in soil for 17 months (Talbert and Fletchall, 1964).

AZIDE

Fishes

The 48-hour LC_{50} for bluegill exposed to azide (potassium) and azide (sodium) was 1,600 and 880 ppb, respectively (FWPCA, 1968).

Arthropods

The LC_{50} for various arthropods to azide is found in table 56.

TABLE 56. The LC_{50} for various arthropods to azide

Formulation	Arthropod Species		Exposure time hr.	LC_{50} ppb.	Source
Sodium	Stonefly	(*Pteronarcys californica*)	24	16	Sanders and Cope, 1968
Potassium	"	(*P. californica*)	96	22	"
Sodium	Amphipod	(*Gammarus lacustris*)	96	8	FWPCA, 1968
Potassium	"	(*G. lacustris*)	96	10	"

BARBAN

Mammals

The LD$_{50}$ for the rat was 1,350 mg/kg to barban when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 24-hour LC$_{50}$ for harlequin fish to barban was 1.5 ppm (Alabaster, 1969).

Persistence

Barban applied to soil persisted at detectable levels for about 3 months (Kearney, Nash and Isensee, 1969).

BENAZOLIN

Mammals

The LD$_{50}$ for the rat was 3,000 mg/kg to benazolin when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC$_{50}$ for harlequin fish to benazolin was 108 ppm (Alabaster, 1969).

BENEFIN

Mammals

The LD$_{50}$ for new-born rats was 800 mg/kg to benefin when the mammals were fed the stated dosage orally (USDA, 1967).

Birds

The LD$_{50}$ for young female mallards was >2,000 mg/kg to benefin when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

BORAX

Mammals

The LD$_{50}$ for the rat was 5,560 mg/kg to borax when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 24-hour LC$_{50}$ for rainbow trout to borax was 2,800 ppm (Alabaster, 1969).

BROMOXYNIL

Mammals

The LD$_{50}$ for the rat was 190 mg/kg to bromoxynil when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 24-hour LC$_{50}$ for harlequin fish to bromoxynil (potassium salt) was 64 ppm (Alabaster, 1969).

CACODYLIC ACID

Mammals

The LD$_{50}$ for the rat was 1,350 to 1,400 mg/kg to cacodylic acid when the mammal was fed the stated dosages orally (House et al., 1967).

Fishes

USDI (1966b) reported that cacodylic acid at 40 ppm had no effect on the longnose killifish during a 48-hour exposure.

The LD$_{50}$ for mosquito fish and taillight shiners approach at 1,000 ppm for cacodylic acid (Oliver, Parsons and Huffstutler, 1966). Largemouth bass, fed for 9 weeks on mosquito fish exposed to 1,000 ppm cacodylic acid for 24 hours, appeared to be unaffected by the treatment.

Mosquito fish, largemouth bass, and taillight shiners exposed to concentrations of 100 ppm of cacodylic acid for 72 hours survived well. Some mortality was observed when concentrations reached 631 ppm with an exposure time of 72 hours (Oliver, 1966).

Amphibians

The 48 hour LC_{50} for *Bufo* tadpoles was between 100 and 1,000 ppm cacodylic acid (Oliver, Parsons and Huffstetler, 1966).

Molluscs

The exposure of the eastern oyster to 40 ppm of cacodylic acid for 48 hours had no noticeable effect (USDI, 1966b).

Arthropods

Cacodylic acid at 40 ppm had no effect on pink shrimp during a 48-hour exposure (USDI, 1966b).

Two species of dragonfly nymphs (*Pantala* sp. and *Gynacantha nervosa*), exposed for up to 72 hours to 1,000 ppm of cacodylic acid, showed no noticeable effects (Oliver, Parsons and Huffstetler, 1966).

Chansler and Pierce (1966) reported that cacodylic acid injected at a rate of 1 to 2 ml per injection at 2-inch intervals around the trunk killed bark beetles (*Dendroctonus adjunctus, D. obesus, D. ponderosae,* and *D. pseudotsugae*). The trees were injected with the herbicide soon after the beetles had attacked the tree and before most of the eggs had hatched. The beetles died before constructing their egg galleries. Some of the eggs failed to hatch, and a high brood mortality occurred. The exact mode of action is not known, but they suspect that the cambium may be killed, causing the death of the beetles, or the herbicide may have direct insecticidal properties.

Plants

The sandhill biotic community underwent significant modification of its flora when cacodylic acid application rates were 6 lb/A or greater (Oliver, Parsons and Huffstetler, 1966). Applications of 30 lb/A to hammock communities killed or defoliated all the exposed plants. Grassland plots treated with 30 lb/A, however, did show some regrowth and recovery after 4 weeks. Algal productivity was reduced in the aquatic habitats at concentrations of cacodylic acid above 2 lb/A.

Persistence

Cacodylic acid appears to break down rapidly within the soil (House et al., 1967 and WSA, 1967). No time was given and the breakdown products were not listed.

CDAA

Mammals

The LD_{50} for the rat was 750 mg/kg to CDAA when the mammal was fed the stated dosage orally (WSA, 1967).

Microorganisms

At normal application rates (4 to 5 lb/A for most uses in WSA, 1967) CDAA reduced soil nitrification based on laboratory tests (Otten, Dawson and Schreiber, 1957). However, CDAA did not affect the microorganisms (*Streptomyces*) when applied at rates from 6 to 300 lb/A (Bounds and Colmer, 1964).

Persistence

CDAA applied at 8 lb/A persisted at detectable levels in soil for 6 weeks (Gantz and Slife, 1960).

At rates of 4 to 5 lb/A CDAA persisted at detectable levels in soil for 3 to 6 weeks, with the longer persistence in the heavier soils (WSA, 1967).

CDEC

Mammals

The LD_{50} for the rat was 850 mg/kg to CDEC when the mammal was fed the stated dosage orally (USDI, 1970b).

Microorganisms

CDEC at normal application rates (2 to 6 lb/A in WSA, 1967) did not affect soil nitrification based on laboratory tests (Otten, Dawson and Schreiber, 1957).

Persistence

CDEC applied at 6 lb/A persisted at detectable levels in soil for 6 weeks (Gantz and Slife, 1960).
At 4 lb/A CDEC persisted at detectable levels for about 3 to 6 weeks in soil, depending upon soil type and rainfall (WSA, 1967).

CHLOREA

Fishes

The 24-hour LC_{50} for rainbow trout to Chlorea was 1,150 ppm (Alabaster, 1969).

CHLORFLURAZOLE

Fishes

The 24-hour LC_{50} for rainbow trout to chlorflurazole was 0.15 ppm (Alabaster, 1969).

CHLOROXURON

Mammals

The LD_{50} for the rat was 3,700 mg/kg and for the dog, >10,000 mg/kg to chloroxuron when the mammals were fed the stated dosages orally (WSA, 1967).

Birds

The LD_{50} for young mallards was >2,000 mg/kg to chloroxuron when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The LC_{50} for killifish to chloroxuron was >50 ppm (WSA, 1967). No exposure time was given. Chloroxuron at 0.4 ppm did not cause mortality in fathead minnows and had no effect on their reproduction at 8 weeks after treatment (WSA, 1967).

CHLORPROPHAM

Mammals

The LD_{50} for the rat was 1,500 mg/kg to chlorpropham when the mammal was fed the stated dosage orally (USDI, 1970b).

Birds

The LD_{50} for young mallards was >2,000 mg/kg to chlorpropham when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Fishes

Davis and Hardcastle (1959) found that bluegill exposed in Ouachita River water had a 24-hour LC_{50} of 10 ppm to chlorpropham. In a later study, Hughes and Davis (1962) reported a 24-hour LC_{50} for bluegill to chlorpropham at 20.0 ppm (liquid formulation) and 10.0 ppm (granular formulation).

Annelids

Chlorpropham applied at 1.8 lb/A had no effect on *Allolobophora caliginosa*, but destroyed 50 percent of *Lumbricus castaneus* (earthworms) (Van der Drift, 1963).

Plants

Sublethal dosages (1 lb/A) of chlorpropham applied to the weed species *Eupatorium maculatum* and *Impatiens biflora* caused an increase in the nitrate content of these plants by 62 percent (8.9 mg/g dry weight to 14.4 mg/g) and 80 percent, respectively (Frank and Grigsby, 1957). These high nitrate concentrations were sufficient to cause nitrate poisoning in livestock if consumed in large enough quantities. In contrast, the chlorpropham treatment caused a 1- to 63-percent reduction in nitrate content of 4 other species of weeds.

Microorganisms

Based on laboratory tests, at normal application rates (2 to 8 lb/A in WSA, 1967) chlorpropham reduced soil nitrification (Otten, Dawson and Schreiber, 1957), and chlorpropham at 50 ppm completely stopped the growth of nitrifying microorganisms (Hale, Hulcher and Chappell, 1957).

The herbicide chlorpropham, applied at rates exceeding 9.0 to 14.4 lb/A, reduced the activity of ammonifying and nitrifying bacteria and the number of *Azotobacter* and *Clostridium pasteurianum* in the soil (Geller and Khariton, 1961). However, in another study chlorpropham applied at 6 to 300 lb/A did not affect the soil microorganisms (*Streptomyces*) (Bounds and Colmer, 1964).

Chlorpropham at normal application rates did not change nitrification in soil (Balicka and Sobieszczanski, 1969a in Balicka, 1969); however, the number of *Azotobacter* did increase in the treated soil (Balicka, 1969).

It was interesting to note that cellulose decomposition in soil was impaired by chlorpropham (Sobieszczanski, 1969 in Balicka, 1969).

Persistence

Chlorpropham applied at 4 ppm persisted at detectable levels in soil for 7 weeks (Burschel and Freed, 1959).

Chlorpropham applied at a rate of 4 to 6 lb/A to moist loam soil persisted for 3 to 5 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

CHLORTHIAMID

Mammals

The LD_{50} for the rat was 757 mg/kg to chlorthiamid when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to chlorthiamid (91 percent) was 41 ppm (Alabaster, 1969).

4-CPA

Fishes

The 24-hour LC_{50} for harlequin fish to 4-CPA was 90 ppm (Alabaster, 1969).

Persistence

4-CPA applied at 25 ppm persisted at detectable levels in soil for 27 days (Burger, MacRae and Alexander, 1962).

CYTROL AMITROLE-T

Fishes

Cope (1965) reported that the 96-hour LC_{50} for bluegills to Cytrol Amitrole-T was 10,000 ppm; and Swabey and Schenk (1963) reported that the 24-hour LC_{50} for Lake Emerald shiner in medium-hard water was 455 ppm to Cytrol Amitrole-T.

Plants

Vance and Smith (1969) reported that Cytrol Amitrole-T at dosages of 150 to 200 ppm inhibited the growth of 3 species of algae from 30 to 76 percent. *Chlamydomonas eugametos* appeared to be less sensitive to Cytrol Amitrole-T than *Scenedesmus quadricauda* and *Chlorella pyrenoidosa*.

Persistence

Cytrol Amitrole-T applied at 2 lb/A was found in streamwater at 400 ppb immediately after spraying (Norris, 1967). Within 10 hours the concentration had dropped to less than 4 ppb; no Cytrol Amitrole-T was detectable 3 days after treatment.

2,4-D

Mammals

The LD_{50} for the rat was 666 mg/kg; for the mouse, 375 mg/kg; for the rabbit, 800 mg/kg; for the dog, 100 mg/kg; and for the guinea pig, 1,000 mg/kg to 2,4-D when the mammals were fed the stated dosages orally (Spector, 1955). The LD_{50} of 2,4-D for mule deer was given at 400 to 800 mg/kg when fed orally in capsules (Tucker and Crabtree, 1970).

Corn plants grown in soil treated with 2,4-D as a pre-emergent treatment (1 to 3 lb/A) were more attractive (about 33 percent more were destroyed) to mice than plants grown in untreated soils (Raleigh and Patterson, 1948). Most of the plants at the time of injury were in the three-leaf stage and about 3 inches tall, but only the kernel of the dug plants was eaten.

Vegetation treated with 2,4-D (alkanolamine salt) in a 5-percent solution repelled cattle (Grigsby and Farwell, 1950).

2,4-D has been reported to repel some mammals (Richter, 1952 in Springer, 1957). For example, cottontail rabbits given a choice of feeding on 2,4-D-treated vegetation or untreated vegetation ate almost none of the treated vegetation.

Treating vegetation with herbicides may alter the plant species composition, and thus the suitability of the habitat for certain mammals. For example, spraying mountain rangeland in Colorado with 2,4-D resulted in several changes in the normal vegetational types and, in turn, in the mammals after one year: (1) the production of perennial forbs was reduced 83 percent, and grass production increased 37 percent after treatment; (2) the diet of pocket gophers changed from 82-percent forbs to 50-percent forbs, and changed from 18-percent grass to 50-percent grass; and (3) the pocket gopher population was reduced 87 percent (Keith, Hansen and Ward, 1959). The suggested reasons for the decline in number of gophers were a depletion in the amount of essential food plants and nitrate poisoning.

In an investigation of the effect of sagebrush control by 2,4-D on use of vegetation by cattle and wildlife in Colorado, Anderson (1960) reported few changes in animal use during the short period of one year. He reported a small decrease in deer use in some of the treated areas. Anderson recommended that to be able to evaluate fully the effect of sagebrush eradication on deer, sage grouse, rabbits, and other animals, the investigation be carried on for several years.

2,4-D and 2,4,5-T at 4, 8, 12, and 16 lb/A improved deer browse by killing off tops of the taller trees and stimulating regrowth at the bases (Krefting and Hansen, 1963). Both herbicides proved most effective at the 12-lb dosage, and 2,4-D was significantly better than 2,4,5-T. The deer showed no preference for either untreated or herbicide-stimulated branch growth.

Lundholm (1970) reported that about 40 percent of a reindeer herd of 600 died in April and May, 1970, when they fed on coniferous vegetation which had been treated on July 12, 1969, with a mixture of 2,4-D (2 parts) and 2,4,5-T (1 part) at a rate of about 2.5 lb/A. Also, 40 of the reindeer aborted their young. Analyses revealed that the coniferous leaves from the area during April and May contained 30 ppm of 2,4-D and 10 ppm of 2,4,5-T.

Birds

The LD_{50} for young mallards was >>1,000 (acid) mg/kg; for young mallards, >>2,025 (sodium salt) mg/kg; for young pheasants, 472 (acid) mg/kg; for young coturnix, 668 (acid) mg/kg; and for pigeons (*Columba livia*), 668 (acid) mg/kg to 2,4-D when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; for bobwhites, >5,000 ppm; and for coturnix, >5,000 ppm of 2,4-D (BEE and dimethylamine salt) in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

2,4-D influenced egg production in chickens exposed for 14 days to grass sprayed daily with 2,4-D (82-percent acid) at ½ oz/gal of water and 2½ oz/gal (Dobson, 1954). The lower 2,4-D treatment led to a 22-percent reduction in egg yield and the higher dosage, to only an 8-percent reduction, but there was no change in the fertility or hatchability of the eggs, nor did the chickens lose any weight. 2,4-D was also found in one test to depress total reproduction of mallard ducks when fed daily at rates of 1,250 and 2,500 ppm, and in another test at the same dosages reproduction was about 80-percent suppressed (USDI, 1970a).

Wild turkeys used the treated right-of-way areas (2,4-D and 2,4,5-T) (Bramble and Byrnes, 1958). The young turkeys were attracted to the openings to feed on various insects more abundant on the grassy right-of-way than within the wooded areas.

Fishes

See table 57 for the LC_{50} for various fish to 2,4-D.

Spot were able to survive a 48-hour exposure to 50 ppm of 2,4-D without any deleterious effect (Butler, 1965).

In laboratory experiments 2,4-D was not toxic to bluegill or largemouth bass at 1 ppm, and only slightly toxic at 100 ppm (King and Penfound, 1946). Then Hiltibran (1967) reported that bluegill, green sunfish, and smallmouth bass fry survived a concentration of 10 ppm of 2,4-D (ethylhexy ester) for 8, 4, and 5 days, respectively, in an experiment lasting 8 days.

In India 2,4-D applied at a rate of 2.5 percent in 100 gallons/A killed 5 percent of the tadpoles, 1.6 percent of the Rahu fish fry, and 3.2 percent of the katla fish fry (Sen, 1957). However, no mortality was observed in native fish in an east-coast estuary when 2,4-D was applied at a rate of 30 lb/A (Beaven, Rawls and Beckett, 1962). Rawls in later investigations (1965) found that 2,4-D acetamide applied to an estuary at 20 lb/A killed all the caged fish (mostly pumpkinseed) within 60 days. 2,4-D butyl ester (BE) or isooctyl ester (IOE) formulations caused little or no mortality to the fish, and these formulations were judged as safe for use against milfoil in marshes.

Mortality among bluegills ranged from 10 to 100 percent in ponds treated with 10 ppm of 2,4-D (Wallen, 1968). Spawning was delayed for 2 weeks in the ponds with 10 ppm; however, fry production appeared to be essentially the same at 10, 5, 1, 0.5, and 0.1 ppm of 2,4-D.

Bluegills were found to convert the herbicide 2,4-DB to 2,4-D (Gutenmann and Lisk, 1965).

Young silver salmon when exposed to a combination of 2,4-D and 2,4,5-T (about 10 percent of each chemical in the combined formulation) at concentrations of 50 ppm or more were observed to be "immediately distressed and would snap their jaws, dart about the aquarium, and leap out of the water before loss of equilibrium and death" (Holland et al., 1960).

The LC_{50} for bluegill to 2,4-D formulations is presented in table 58 (Hughes and Davis, 1963). The ester formulations appeared to be most toxic to the fish, probably due to more effective penetration. Hughes and Davis did not attempt to explain the wide variation in results obtained from the different batch lots of the same formulation.

A group of experimental ponds were treated with 2,4-D at concentrations of 0.1, 0.5, 1, 5, and 10 ppm (Cope, Wood and Wallen, 1970). About 10 percent of the bluegills died within 8 days with

TABLE 57. The LC_{50} for various fish to 2,4-D.

Formulation	Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Butyl Ester	Harlequin fish	24	1.0	Alabaster, 1969
Oleic-1,3-propylene diamine	Bluegill	24	4.0	Davis and Hughes, 1963
Butyl Ester	Bluegill	24	4.9	"
Butyl Ester	Bluegill	24	10	"
	Rainbow trout	24	250	Alabaster, 1956
Amine	Rainbow trout	24	250	Alabaster, 1969
Ethylhexy Ester	Lake Emerald shiner	24	200	Swabey and Schenk, 1963
Ethylhexy Ester	Lake Emerald shiner	24	620	"
Sodium Salt	Harlequin fish	24	1,160	Alabaster, 1969
Isopropyl	Bluegill	48	0.8	FWPCA, 1968
Propylene Glycol Butyl Ether Ester	Rainbow trout	48	0.96	"
	Rainbow trout	48	1.1	Bohmont, 1967
Butyl Ester	Bluegill	48	1.3	FWPCA, 1968
Mixed Butyl and Isopropyl Esters	Bluegill	48	1.5	"
Butoxyethanol Ester	Bluegill	48	2.1	"
	Bluegill	48	5.7	Bohmont, 1967

the highest concentration. At 5 ppm and below mortality was negligible. Growth in weight was nearly 3 times that of the control fish in the pond treated with 10 ppm of 2,4-D. Growth at the other 2,4-D concentrations was also greater than in the control, but not as great as in the 10 ppm concentration. The most severe pathologic lesions were observed in fish at the highest concentrations, and this lasted for nearly 84 days. The pathologic effects involved the liver, vascular system, and brain.

TABLE 58. The LC_{50} for bluegill to 2,4-D formulations, including different batches of same formulation (Hughes and Davis, 1963).

Formulation	LC_{50} (Acid Equiv. ppm) 24 hr	48 hr
2,4-D		
Alkanolamine, ethanol and isopropanol series	900	840
Alkanolamine, ethanol and isopropanol series	588	530
Alkanolamine, ethanol and isopropanol series	450	435
Dimethylamine	542	458
Dimethylamine	500	416
Dimethylamine	390	353
Dimethylamine	375	375
Dimethylamine	220	220
Dimethylamine	166	166
Di-N,N-dimethylcocoamine	1.5	1.5
2,4-D acid, with emulsifiers	8.0	8.0
Isooctyl ester	66.5	56.7
Isooctyl ester	36.0	36.0
Isooctyl ester	8.8	8.8
Propylene glycol butyl ether ester	2.1	2.1
Butoxyethanol ester	2.1	2.1
Butyl ester	1.3	1.3
Mixed butyl and isopropyl esters	1.7	1.7
Mixed butyl and isopropyl esters	1.6	1.5
Isopropyl ester	0.9	0.8
Ethyl ester	1.4	1.4

In laboratory experiments conducted by Mr. Jack Lowe, fish were exposed to 2,4-D and carbaryl (no dosage given) for 1 to 5 months ([illegible], 1969). The exposed fish grew as well as the controls and had little mortality; however, careful examination revealed massive invasions of the central nervous system of the test fish by what appeared to be a microsporidian parasite. The author suggested that the pesticides lowered the natural resistance of the fish to parasite attack.

An investigation of the persistence of 2,4-D in fish revealed that 50 percent of the chemical was lost in <1 week (Macek, 1969).

Amphibians

At 0.5-percent solution 2,4-D was found to inhibit the development of frog (*Rana temporaria*) eggs (Lhoste and Roth, 1946).

The 24-hour LC_{50} for chorus frog tadpoles exposed to 2,4-D was 100 ppm (Sanders, 1970).

Molluscs

The exposure of eastern oysters to 2.0 ppm of 2,4-D acid for 96 hours had no effect on shell growth (Butler, 1963).

Two weeks after an estuary in Virginia was treated with 2,4-D at a rate of 10 lb/A for milfoil (Haven, 1963), there was a significant reduction in the numbers of a small mollusc (*Macoma baltica*), and the population remained low for some 8 months. Haven felt that the reduction of the mollusc population was due to the decay of the milfoil and associated anaerobic conditions, and not directly to the herbicide.

Field application of 2,4-D with dosages as high as 120 lb/A did not affect caged eastern oysters and clams in the treated areas (USDI, 1962). Beaven, Rawls and Beckett (1962) also found 2,4-D safe for eastern oysters and soft-shell clams when applied at a rate of 30 lb/A. However, Rawls (1965) found that 2,4-D acetamide applied to an estuary at 20 lb/A killed all the caged eastern oysters and soft-shell clams within 30 days. But Rawls did find that 2,4-D BE and IOE formulations were safe for these molluscs.

Arthropods, Annelids, and Other Invertebrates

The LC_{50} for 2,4-D tested against the fiddler crab (*Uca pugnax*) at different times and dosages was as follows: 5,000 ppm for 96 hours, 2,500 ppm for 10 days, and 1,000 ppm for 17 days (Sudak and Claff, 1960).

The LC_{50} for various arthropods to 2,4-D is found in table 59.

The minimum lethal concentrations (ppm) of 2,4-D which produced a kill of fish-food organisms exceeding 25 percent are the following: *Daphnia*, 0.2; *Eucypris*, 0.6; *Hyalella*, 0.6; *Palaemonetes*,

TABLE 59. The LC_{50} for various arthropods to 2,4-D.

Formulation	Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Butoxyethanol ester	Amphipod (*Gammarus lacustris*)	24	1.4	Sanders, 1969
Propylene glycol butylester	" (*G. lacustris*)	24	2.1	"
Isooctyl ester	" (*G. lacustris*)	24	0.6	"
Butyl ester	Stonefly (*Pteronarcys californica*)	24	0.5	Sanders and Cope, 1968
	" (*P. californica*)	24	50	"
Dimethylamine	Amphipod (*G. lacustris*)	24	>100	Sanders, 1969
Butoxyethanol ester	Stonefly (*P. californica*)	48	1,800	FWPCA, 1968
Propylene glycol butylether ester	Amphipod (*G. lacustris*)	48	1,800	"
Propylene glycol butyl ether ester	Waterflea (*Daphnia pulex*)	48	3,200	"

0.8; *Amphiagrion*, 5.0; *Pachydiplax* and *Tramea*, 4.5; *Culex*, *Aedes*, and *Anopheles*, 5.5; *Chironomus*, 1.0; *Physa*, 5.5; and *Helisoma*, 7.5 (Ziuchkalv, 1952).

Brown shrimp exposed to 2.0 ppm of 2,4-D for 48 hours showed a 10-percent mortality or paralysis (Butler, 1965). The median immobilization concentration of 2,4-D to *Daphnia magna* was found to be 100 ppm (Crosby and Tucker, 1966). This concentration for *D. magna* is about 500 times greater than that given for *Daphnia* above. There is no explanation given.

Two weeks after an estuary in Virginia was treated for milfoil control with 2,4-D at 10 lb/A, there was a significant reduction in the numbers of an amphipod (*Leptocherius plumulosus*) (Haven, 1963), and the population remained low for some 5 months (see comment under Molluscs).

No mortality was observed in native crabs exposed to 2,4-D applied at a rate of 30 lb/A (Beaven, Rawls and Beckett, 1962). Furthermore, field applications of 2,4-D as high as 120 lb/A did not kill caged blue crabs (USDI, 1962). Rawls (1965), however, found that 2,4-D acetamide applied to an estuary at 20 lb/A killed all the caged blue crabs within 30 days. But the 2,4-D BE or IOE formulations caused little or no mortality to the test animals, and these formulations were judged safe for use against milfoil in the marshes.

2,4-D applied at rates of 20 and 40 lb/A did not significantly influence the numbers or weight of bottom invertebrates (Hooper, 1958). However, Walker (1962) reported the following bottom organisms were reduced 50 percent or more one week after treatment with 2,4-D (1 to 4 ppm): mayfly nymphs, horsefly nymphs, common midges, phantom midges, biting midges, caddice fly larvae, water beetles, aquatic worms, and leeches (plus clams and snails). In another investigation no significant changes in numbers of burrowing mayflies (*Hemagenia*) were measured after the treatment of the reservoirs with 100 lb (1 ppb in water) per acre of 2,4-D (Smith and Isom, 1967). The conclusion from these investigations was that low concentrations of 2,4-D (20 to 100 lb/A, resulting in about 1 ppb of 2,4-D) have little effect upon bottom organisms.

Of mosquito larvae treated with 2,4-D at a rate of 100 ppm in water, about three-fifths fewer larvae as in the control reached the pupal state (Smith and Isom, 1967). This study added further evidence that 2,4-D is relatively non-toxic to some invertebrate species.

Water treated with 40 to 100 pounds per acre of 2,4-D for control of water milfoil did not appear to affect the aquatic fauna or water quality (Smith and Isom, 1967).

Jones and Connell (1954) calculated the LD_{50} of 2,4-D fed orally to honeybees at 104.5 μg/bee; however, Beran and Neururer (1955) reported an LD_{50} about 1/10 this level, or 11.520 μg/bee.

Treating fields in New Zealand for ragwort control with 2,4-D at 3 lb/A caused a 22-percent mortality in honeybees working the treated field (Palmer-Jones, 1964). Dusting bees with 2,4-D, however, did not cause any mortality. Palmer-Jones raised the question whether the toxicity observed in the field was due to the 2,4-D dissolved in the nectar or to the production of a toxic metabolite secreted by the plant into the nectar.

2,4-D may also benefit bees, as reported by Bellmann (1950). The herbicide was used to restore bee pasture along the side of the road by destroying brush and other weeds, thus encouraging sweet clover to regain its dominance.

Fox (1964) reported that 2,4-D at 1 and 2 lb/A increased the wireworm (*Ctenicera aeripennis destructor*) damage to wheat. At 1 lb/A 31 percent of the wheat plants were killed, whereas in the untreated check only 5 percent were killed. The exact reason for the increased kill of wheat plants is not known, but one proposed reason was that 2,4-D delayed the growth of plants, thus making them more susceptible to wireworms.

Putnam (1949) reported that the number of grasshoppers (*Melanoplus mexicanus*) per square yard was about double in the 2,4-D (1 lb/A)-treated plots: 59 per sq. yd. in the 2,4-D-treated, compared with 30 per sq. yd. in the check. The indications were that 2,4-D hastened the development or increased the survival of the grasshoppers.

Dipping bean plants into 2,4-D at levels of 4.1 ppm increased aphid progeny production during a 10-day period from 159 to 764 per aphid (Maxwell and Harwood, 1960). In other experiments with a high dosage of 41.0 ppm of 2,4-D aphid production was less stimulated than at the lower dosage of 4.1 ppm. Some amino acids were at higher levels in the treated plants than in the untreated plants, and this probably improved the food resource for the aphids.

The longevity of aphid adults and the growth rate of grasshopper nymphs appeared to be unaffected by the 2,4-D treatments (Maxwell and Harwood, 1960).

Adams (1960) reported that 2,4-D sprayed at a rate of ½ lb/A on coccinellid beetle larvae (*Coccinella transversoguttata*, *C. perplexa*, and *Hippodamia tredecimpunctata*), especially those in the late larval stages, killed between 70 and 75 percent of the animals. Also, the mean developmental time of the treated larvae increased significantly, in some cases from 16 to 27 days.

Aphids (primarily *Rhopalosiphum padi*) were more abundant on oats in fields treated with 2,4-D at ½ lb/A (Adams and Drew, 1965). Aphid outbreaks occurred, they suggested, because there were fewer coccinellid predaceous beetles present in the treated areas, and the activity of the coccinellids present was depressed.

Rice stem-borer larvae grew almost 45 percent larger (35.1 mg on 2,4-D versus 24.4 mg for the control larvae) during the 30-day experimental period when rice plants on which the larvae fed were treated with 2,4-D (Ishii and Hirano, 1963). But when 2,4-D was added to sterilized rice stems fed to the rice stem-borer, larval growth was not improved. The explanation given by the authors was that 2,4-D increased the nitrogenous level in the growing rice plants, improving the plants as food for the larvae.

No mortality occurred in earthworms when they were immersed for 2 hours in concentrations of 0.1, 1.0, 10.0, and 100 ppm of 2,4-D, but at 1,000.0 ppm 100 percent mortality occurred (Martin and Wiggans, 1959).

2,4-D at normal dosages did not affect the numbers of wireworms, springtails, mites, and other micro-arthropods in soil (Van der Drift, 1963 and Rapoport and Cangioli, 1963).

Red clover plants resistant to the nematode *Ditylenchus dipsaci* lost this resistance when the plants were treated with 2,4-D (Webster and Lowe, 1966). Susceptible clover plants were made more attractive to nematodes after their treatment with 2,4-D. Red clover is not a normal host to the nematode *Aphelenchoides ritzemabosi*, but the nematode fed on the tissues treated with 2,4-D, significantly increasing the nematode's rate of reproduction. Webster and Lowe also found that 2,4-D greatly increased the reproduction of the nematode *A. ritzemabosi* in lucerne callus. They reported that soaking nematodes in 2,4-D solutions up to 5 ppm did not harm them, but that concentrations of 50 ppm did suppress their reproduction.

Spraying 2,4-D at a rate of 140 mg/sq. yd. onto nematode-susceptible and -resistant oats infested with *D. dipsaci* increased the number of nematodes per plant in both the resistant and susceptible cultivars (Webster, 1967). The number of nematodes was at least double in some treatments. Nematodes did not reproduce on the unsprayed resistant oat plants, whereas the nematodes associated with the 2,4-D treated plants produced a large number of eggs. The evidence suggested that in treated plants nematodes infesting the oats from the soil reproduced more than those inoculated directly into the oats.

Plants

Concord grapes were most sensitive to 2,4-D, and quantities as small as 0.0001 μg placed on a young leaf caused a malformation of from 4 to 6 leaves (Clore and Bruns, 1953). However, the exposure of phytoplankton to 1.0 ppm of 2,4-D for 4 hours did not cause a decrease in growth (Butler, 1965).

A significant increase in protein content of the grain was noted in wheat grown in 2,4-D treated plots (Helgeson, 1947). Treatments with 2,4-D also increased the amount of protein in wheat in direct relationship to the amount of 2,4-D used (Erickson, Seely and Klages, 1948). At a dosage of 4.0 lb/A the protein level in wheat was 15.5 percent, whereas in the untreated control the protein level was only 10.9 percent.

Beans grown as a second crop on the previously 2,4-D-treated soil were observed to have a lower level of protein than the control (Anderson and Baker, 1950).

Nitrogen was higher in wheat grain treated with 2,4-D than in untreated grain (Pande, 1954). The increase in nitrogen was associated with increasing concentrations of 2,4-D (3.25 to 6.5 lb/A). It is interesting to note that the nitrogen level in wheat also increased when weeds were hand-pulled from the plots. Fults and Payne (1956) reported that treating bean plants with 2,4-D spray (1,000 ppm) caused a significant decrease in total free amino acids, in contrast to an increase in total amino acid in sugar beet and potato.

Willard (1950) reported that 2,4-D altered the palatability of some plant species to animals, such as livestock eating Canada thistle, velvet-leaf, jimson weeds, wild parsnip, sunflowers, round-leafed mallow, and other unpalatable weeds. The ragwort weed, highly toxic to cattle, had a marked increase in sugar content after treatment with 2,4-D. The high sugar content made the plants attractive to cattle, but they were still highly toxic. Normally, livestock and wildlife will not feed on ragwort unless forced to do so.

A sugar-beet field was treated with a "sublethal" dosage of 2,4-D by mistake (the farmer treated with 2,4-D-contaminated toxaphene), resulting in disfigured plants with a high level of nitrate (Stahler and Whitehead, 1950). The 2,4-D treatment increased the level of potassium nitrate in the beets from 0.22 percent by dry weight to 4.50 percent. A 1.5-percent level is toxic to cattle.

Sublethal concentrations of 2,4-D caused the level of potassium nitrate in Canada thistle and Russian pigweed to double (Berg and McElroy, 1953); in Canada thistle, 1.30 to 2.64 percent by dry weight, and in Russian pigweed, 2.45 to 4.88 percent. Sublethal spray applications of 2,4-D on both mustard and sugar beet plants resulted in increased levels of nitrates in the plants, but the increase was not much above 10 percent in most cases (Whitehead, Kersten and Jacobsen, 1956). Pigweed, lambsquarter (*Chenopodium*), and smartweed (*Polygonum*) were found to have extremely high levels of nitrate after treatment with 2,4-D (Olson and Whitehead, 1940 in Willard, 1950).

After the treatment of 9 species of weeds with sublethal concentrations (0.25 lb/A) of 2,4-D, potassium nitrate content declined from 6 to 44 percent in 5 species and increased from 12 to 47 percent in the other 4 species (Frank and Grigsby, 1957). In *Eupatorium maculatum* the increase was 47 percent. The nitrate levels in these species and several other species of plants were sufficiently high to cause nitrate poisoning in livestock if consumed in large enough quantities.

After buckwheat was sprayed with 50, 100, 500, and 1,000 ppm of 2,4-D the sugar values in the stems and leaves increased and then fell to a very low level by the eighth day after treatment (Wort, 1951). Total nitrogen and protein nitrogen in the stems and roots increased with both time and concentration after the herbicide application.

Black cherry brush was treated until wet with a concentration of 2,4-D at 2,000 ppm (Grigsby and Ball, 1952). By the 15th day after treatment, the hydrocyanic acid (HCN) present in the cherry leaves had been reduced by about 88 percent (control=91.9 mg/100 g fresh wt.; 2,4-D treated=11.5 mg/100 g).

Swanson and Shaw (1954) demonstrated that 2,4-D caused an initial decrease in the quantity of hydrocyanic acid in Sudan grass, but 4 days after treatment there was an increase in HCN over the controls. Results of their tests showed that the hydrocyanic acid content of Sudan grass was increased by 36 percent (control, HCN 30 mg/100 g fresh wt. versus 2,4-D 40 mg/100 g) in plots treated with 1 lb/A of 2,4-D. Note that the LD_{50} for sheep of HCN as a free glucoside is about 4.5 mg/kg body weight (Coop and Blakley, 1950).

Fertig (1953) reported that nitrate content of lambsquarter and pigweed may increase as much as 5.5 percent (dry weight). This would mean that 20 to 25 pounds of fresh green material would be toxic to a 500-pound animal.

Plants receiving high levels of nitrogen were more susceptible to 2,4-D than bean plants on low-N (Freiburg and Clark, 1951). 2,4-D also changed the absorbing capacity of bean-plant roots, as indicated by the failure of the treated plants to increase their content of total nitrogen, including nitrates, after exposure of 2,4-D. The treated bean plants showed a decrease in the percentage of protein and nitrogen in the leaves, but an increase in the stems and roots.

2,4-D applied to irrigation water at rates of 11 ppm caused tomato and cotton plants to grow more vigorously, but injured tokay and Concord grapes (Olsen, 1954).

Spraying 2,4-D and 2,4,5-T herbicides (2 quarts 2,4-D and 1 quart 2,4,5-T per 100 gallons of water) at a height of 4 to 6 feet along a roadway caused extensive damage to white, scarlet, and black oaks, plus other trees and shrubs (Niering, 1959).

Blaisdell and Mueggler (1956) reported that when 2,4-D was used at 1.5 to 2 lb/A on 15 shrubs and trees in the treated area, only serviceberry, threetip sagebrush, and silver sagebrush suffered moderate to heavy mortality. Aerial portions of snowbrush, downy rabbitbrush, aspen, chokecherry, willows (*Salix* sp.), and snowberry were affected, but most of these species sprouted profusely later. Bitterbrush, a valuable forage species, was unharmed or only slightly damaged. These authors point out that because of the differences in response of various associated forbs, shrubs, and trees, vegetational composition should always be considered when planning brush control.

Microorganisms

Worth and McCabe (1948) reported that when 2,4-D solutions of 1 and 2 percent were used in the medium, the herbicide inhibited the growth of the aerobic organisms, but had little effect on the facultative anaerobes. In some instances, the growth of the anaerobes was actually stimulated.

The herbicide 2,4-D did not inhibit ammonifying bacteria at concentrations of 0.25 percent and below (Jones, 1956). This was well below the rate ordinarily added to soil.

2,4-D at concentrations below 1,000 mg/l was observed to have little effect on bacterial growth of *Bacterium lactis aerogenes* (Dean and Law, 1964).

Anderson and Baker (1950) reported some inhibition of microorganisms, especially the gram-positive organisms, in the soil with 2,4-D at normal application rates; however, this inhibition of growth was quite transitory. Beans grown as a second crop on the treated soil had a lower level of protein than the control.

At normal field applications of 2,4-D (1 to 4 lb/A) the herbicide had little effect on soil microorganisms (Kratochvil, 1950; Hoover and Colmer, 1953; Fletcher, 1960; and Rounds and Colmer, 1964).

At a dosage of 50 ppm of 2,4-D nitrification in the soil was completely inhibited (Slepecky and Buck, 1960). After the treatment of the soil with 2,4-D an actinomycete was reported as dominating the soil flora (Warren, Graham and Gale, 1951). The actinomycete had rather strong anti-fungal properties, and thus had an inhibiting effect upon soil fungi.

2,4-D applied in 0.1-, 0.5-, and 1-percent solutions favored the increase of soil microorganisms (Ilin, 1962). The 1-percent 2,4-D caused protozoa to cease their activity in 1 to 2 seconds and form cysts. The increase in the number of soil microorganisms may be due to the inhibition of protozoa in the soil. Rapoport and Cangioli (1963) supported this idea, reporting that using sodium salt of 2,4-D inhibited soil protozoa and resulted in an increase in the number of bacteria. 2,4-D stimulated the growth of saprophytic microorganisms in water (Petruk, 1964).

At 10 and 50 ppm of 2,4-D, *Aspergillus niger* proliferation was significantly limited (Arnold, Santelmann and Lynd, 1966). *A. niger* degraded 2,4-D faster than it did picloram, which had little effect upon the fungus.

Biological Concentration

The eastern oyster concentrated 0.1 ppm of butoxy-ethanol ester of 2,4-D in water to a level of 18.0 in itself during 7 days, as measured by 2,4-D acid (Butler, 1965). When a sample of these oysters was placed in clean water for 7 days, the 2,4-D disappeared from the bodies of the oysters.

Esters of 2,4-D accumulated in sunfish after exposure to sublethal concentrations in both laboratory and field tests (Cope, 1965b), and the fish sampled from a reservoir with 1 ppb showed an uptake of 2,4-D to a maximum of 150 ppb (Smith and Isom, 1967).

Within an hour after being treated with 2,4-D at a rate of 100 lb/A, the concentration of 2,4-D in reservoir water was about 1 ppb (Smith and Isom, 1967). Mussels (primarily *Elliptio crassidens*) exposed to the water for 96 hours concentrated the 2,4-D; 2 samples of mussels had an average of 580 ppb and 700 ppb of 2,4-D in their tissues. Asiatic clams concentrated 2,4-D to < 140 ppb.

Resistance

Abel (1954) has noted that increasing doses of 2,4-D were required to control creeping thistle, which suggests the development of resistance in this weed.

There was evidence that strains of broad-leaved plants with a relatively high inherited tolerance for 2,4-D have been selected chemically in the spraying process in sugar-cane fields since 1945 (Hanson, 1956).

Persistence

The evidence suggests that under normal use 2,4-D persists in soil for about a month. In moist loam soil 2,4-D applied at a rate of ½ to 8 lb/A persisted for 1 to 4 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961). Sheets and Harris (1965) reported also that 2,4-D at normal recommended dosages persisted for about 1 month in the soil.

2,4-D and 2,4,5-T was applied as a 1 : 1 mixture as low volatile esters in diesel oil at rates of 2 lb/A (Norris, 1967). From a maximum of 76 ppb, the residue dropped rapidly to less than 0.5 ppb a few days after spraying. Four gallons of diesel oil per acre had little or no effect on the decomposition of 2,4-D in forest litter; in other tests, DDT applied at 1 lb/A appeared to stimulate the breakdown of 2,4-D (Norris and Greiner, 1967).

2,4-D appears to degrade rather rapidly in water. For example, the concentration dropped from 1,000 ppm of application rate to 10 ppb within 80 days (House et al., 1967). However, significant concentrations of 2,4-D (58.8 ppm) were recorded and isolated from sediment samples removed from a reservoir some 10 months after treatment (Smith and Isom, 1967).

From 1 to 2 percent of the 2,4-D applied at a rate of 689 ppb to 967 ppb to water, remained for 81 days after treatment (Averitt, 1967). The most rapid decline, occurring about 4 days after application in the 2 lagoons treated, was not due to heavy rainfall. The author could give no explanation for this rapid loss.

2,4-D applied at 4 lb/A persisted in soil for 4 to 16 weeks (Hernandez and Warren, 1950).

At 10 ppm 2,4-D was found to persist in ponds in Oklahoma for 6 weeks, although in bluegill fish none was detected after 4 days (Cope, Wood and Wallen, 1970).

DALAPON

Mammals

The LD_{50} for the rat was 7,570 to 9,330 mg/kg; for the female mouse, >4,600 mg/kg; for the female rabbit, 3,860 mg/kg; and for the female guinea pig, 3,860 mg/kg to dalapon when the mammals were fed the stated dosages orally (WSA, 1967).

Birds

Several species of birds survived when their diet contained as much as 5,000 ppm of various herbicides. Dalapon was found to depress reproduction in mallard ducks when fed at levels of less than 25 percent of those which produced mortality (USDI, 1962).

The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; and for coturnix, >5,000 ppm of dalapon in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

Fishes

When Lake Emerald shiners were exposed for 3 days to 5,000 ppm of dalapon, no adverse effects were observed (Springer, 1957). See table 60 for LC_{50} for various fish to dalapon. There appears to be some discrepancy in the toxicity of dalapon to bluegills.

Longnose killifish exposed to 1.0 ppm of dalapon (sodium salt) for 48 hours exhibited no noticeable effects (Butler, 1963).

Hiltibran (1967) reported that bluegill, green sunfish, lake chub-sucker and smallmouth bass fry survived a concentration of 50 ppm of dalapon for 8 days or the termination of the experiment.

Molluscs

The exposure of the eastern oyster to 1.0 ppm of dalapon (sodium salt) for 96 hours had no noticeable effect on shell growth (Butler, 1963).

TABLE 60. The LC_{50} for various fish to dalapon

Formulation	Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
	Bluegills	96	115	Cope, 1965a
Sodium	Harlequin fish	[1] 96	300	Alabaster, 1969
Sodium	Rainbow trout	[1] 96	340	"
	Fathead minnow	[1] 96	440	Surber and Pickering, 1962
	Bluegills	[1] 96	480	"
	Rainbow trout	96	>100	Alabaster, 1969
	Harlequin fish	24	>100	"
	Bluegills	48	115	Bohmont, 1967
	Salmon	48	340	"

[1] In soft water

Arthropods, Nematodes, and Annelids

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) exposed to dalapon was 6,000 ppb (FWPCA, 1968).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *D. pulex*, to dalapon was 16 ppm and 11 ppm respectively (Sanders and Cope, 1966).

Stonefly nymphs (*Pteronarcys californica*) exposed to dalapon for 96 hours at 100 ppm were not affected (Sanders and Cope, 1968).

Courtney, Peabody and Austenson (1962) found that dalapon applied at 5 lb/A to colonial bentgrass reduced the number of nematodes by 94 percent.

The exposure of brown shrimp to 1.0 ppm of dalapon for 48 hours cause a 48-percent mortality or paralysis (Butler, 1962).

Dalapon at normal application dosages (0.75 to 20 lb/A in WSA, 1967) was observed to increase the numbers of millipedes, springtails, and mites in soil, but did not cause any significant change in the numbers of earthworms (Fox, 1964).

The 24-hour LC_{50} for stonefly nymphs (*Pteronarcys*) to dalapon (sodium salt) was 1.0 ppm (Cope, 1965a).

Microorganisms

The presence of amitrole inhibited the microbial degradation of dalapon (Kaufman, 1966). The phytotoxic residues of both dalapon and amitrole persisted in the soil longer when the 2 chemicals were applied in combination than when applied separately. Dalapon, especially, disappeared more slowly when amitrole also had been applied to the soil.

Persistence

Dalapon applied at a rate of 5 to 40 lb/A to moist-loam soil persisted for 10 to 60 days with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

Dalapon applied at 50 ppm persisted in soil for < 2 to > 8 weeks (Day, Jordan and Russel, 1963).

DCPA

Mammals

The LD_{50} for rats was 3,000 mg/kg to DCPA when the mammals were fed the stated dosage orally (FCH, 1970).

Plants

Vance and Smith (1969) report that DCPA inhibited the germination of seeds of certain higher plants, but at concentrations up to 200 ppm DCPA showed no toxic effects on any of the algae *Scenedesmus quadricauda*, *Chlamydomonas eugametos*, and *Chlorella pyrenoidosa*.

DEF

Mammals

The LD_{50} for rats was 880 mg/kg to DEF when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

When chickens were fed DEF at a dosage of 900 mg/kg, the chickens developed leg weakness (Gaines, 1969).

Fishes

The 48-hour LC_{50} for bluegill exposed to DEF was 86 ppb (*FWPCA*, 1968).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles exposed to DEF was 1.9 ppm (Sanders, 1970).

Arthropods

The LC_{50} for various arthropods to DEF is found in table 61.

Table 61. The LC_{50} for various arthropods to DEF.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	0.380	Sanders, 1969
Stonefly (*Pteronarcys californica*)	24	3.5	Sanders and Cope, 1968
Amphipod (*G. lacustris*)	48	0.320	FWPCA, 1968
Stonefly (*P. californica*)	48	2.3	"

DIALLATE

Mammals

The LD_{50} for the rat was 395 mg/kg to diallate when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 24-hour LC_{50} for harlequin fish to diallate was 12 ppm (Alabaster, 1969).

DICAMBA

Mammals

The LD_{50} for the rat was 2,900 mg/kg to dicamba when the mammal was fed the stated dosage orally (USDI, 1970b).

Birds

The LD_{50} for dicamba when tested against pheasants was 673 mg/kg (female) and 800 mg/kg (male) (Edson and Sanderson, 1965).

Fishes

The 24-hour LC_{50} for dicamba tested against juvenile coho salmon was 151 ppm (Bond, Fortune and Young, 1965). The estimated 48-hour LC_{50} for dicamba was 35 ppm for rainbow trout and 130 ppm for bluegills (Bohmont, 1967).

Arthropods

The 24-hour LC_{50} for an amphipod (*Gammarus lacustris*) exposed to dicamba was 10,000 ppb (Sanders, 1969).

The 48-hour LC_{50} for amphipods (*G. lacustris*) exposed to dicamba was 5,800 ppb (*FWPCA*, 1968).

Honeybees appear to be extremely sensitive to dicamba; the LD_{50} of dicamba fed to bees was measured at 3.6 µg/bee (Edson and Sanderson, 1965).

Persistence

Dicamba applied to soil persisted for about 3 months (Kearney, Nash and Isensee, 1969).

DICHLOBENIL

Mammals

The LD_{50} for the rat was 3,160 mg/kg to dichlobenil when the mammal was fed the stated dosage orally (USDI, 1970b).

Birds

The LD_{50} for young mallards was >2,000 mg/kg and for pheasants, 1,189 mg/kg to dichlobenil when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for pheasants was 1,500 to 2,500 ppm and for coturnix, >5,000 ppm of dichlobenil in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970).

Fishes

The LC_{50} for various fish to dichlobenil is found in table 62.

Hiltibran (1967) reported that green sunfish, lake chub-sucker, and smallmouth bass fry survived a concentration of 20 ppm of dichlobenil for 8 days or the termination of the experiment; at 10 ppm bluegill fry also survived the 8-day exposure period.

TABLE 62. The LC_{50} for various fish to dichlobenil.

Formulation	Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Wettable powder	Bluegill	24	17	Hughes and Davis, 1962
	Bluegill	24	23	Cope, 1965a
	Rainbow trout	24	23	"
Granular	Bluegill	24	27	Hughes and Davis, 1962
	Harlequin fish	24	120	Alabaster, 1969
	Redear	48	>20	Cope, 1969
	Rainbow trout	48	20.0	Bohmont, 1967
	Bluegill	48	20.0	"

Dichlobenil applied at 10, 20, and 40 ppm to small ponds affected the survival and growth of the fish fauna (mostly bluegills with limited numbers of green sunfish, largemouth bass, and yellow perch) (Cope, McCraren and Eller, 1969). After 100 days survival in the control ponds was 60 percent, at 10 ppm survival was 32 percent, at 20 ppm survival was 6 percent, and at 40 ppm survival was 3 percent. Growth, on the other hand, was greater in the treated ponds. Percent increases were as follows: control, 67 percent; 10 ppm, 169 percent; 20 ppm, 180 percent; and 40 ppm, 482 percent.

Arthropods

The LC_{50} for various arthropods to dichlobenil is found in table 63.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to dichlobenil was 5,800 ppb and 3,700 ppb, respectively (Sanders and Cope, 1966).

The median immobilization concentration of dichlobenil to *Daphnia magna* was found to be 9.8 ppm (Crosby and Tucker, 1966).

Biological Concentration

Esters of dichlobenil accumulated in sunfish after exposure to sublethal concentrations in both laboratory and field tests (Cope, 1966b).

TABLE 63. The LC_{50} for various arthropods to dichlobenil.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	16	Sanders, 1969
Stonefly (*Pteronarcys* sp.)	24	43	Cope, 1965a
" (*P. californica*)	24	49	Sanders and Cope, 1968
Amphipod (*G. lacustris*)	48	1.5	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	3.7	"
Stonefly (*P. californica*)	96	8.4	"

Persistance

Dichlobenil applied at 4 lb/A persisted in soil for 10 months (Niagara Chemical Div., 1961).

Dichlobenil applied at a rate of 4 lb/A two years earlier was still found at levels of 0.19 ppm in the soil of cranberry bogs (Miller, Demoranville and Charig, 1966). The chemical did not leach downward in the soil. Application of a granular formulation of dichlobenil at 0.6 ppm to a farm pond produced the highest residues in water and fish about 2 weeks after the treatment, whereas samples of vegetation and soil had the highest levels within 1 to 2 days (Van Valin, 1966). Residues were still measurable some 188 days later.

An investigation of the persistence of dichlobenil in fish revealed that 50 percent of the chemical was lost in <2 weeks (Cope, McCraren and Eller, 1969).

Dichlobenil applied as a 50-percent wettable powder at rates of 10, 20, and 40 ppm to small ponds disappeared rapidly (Cope, McCraren and Eller, 1969). Only about 8 percent remained after 11 days, and none was detected after 55 days. However, when dichlobenil was applied as a 4-percent granular formulation at a rate of 58 ppb, even at 189 days 1 ppb of dichlobenil was found in the water.

Dichlobenil applied to soil persisted for about 4 months (Kearney, Nash and Isensee, 1969).

DICHLORPROP

Mammals

The LD$_{50}$ for mice was 400 mg/kg to dichlorprop when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 48-hour LC$_{50}$ for bluegill exposed to dichlorprop was 1,100 ppb (FWPCA, 1968).

Persistance

Dichlorprop applied at 25 ppm persisted in soil for >103 days (Burger, MacRae and Alexander, 1962).

DIQUAT

Mammals

The LD$_{50}$ for the rat was 400 to 440 mg/kg to diquat when the mammal was fed the stated dosage orally (WSA, 1967).

Birds

The LD$_{50}$ for young mallards was 564 mg/kg to diquat when the ducks were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970). The LC$_{50}$ for mallards was >5,000 ppm; for pheasants, 3,600 to 5,000 ppm; and for coturnix, 1,400 to 1,600 ppm of diquat in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

Fishes

The LC$_{50}$ for various fish to diquat is found in table 64.

Longnose killifish exposed to 1.0 ppm of diquat for 48 hours showed no noticeable effects (Butler, 1965).

Hiltibran (1967) reported that bluegills, lake chubsuckers, and smallmouth bass fry survived a concentration of 2.5 ppm of diquat (cation) for 8, 9, and 1 days, respectively.

An investigation of the persistence of diquat in fish revealed that 50 percent of the chemical was lost in <3 weeks (Macek, 1969).

Molluscs

The exposure of eastern oysters to 1.0 ppm of diquat of 96 hours had no noticeable effect on shell growth (Butler, 1965).

Arthropods and Annelids

After the destruction of aquatic vegetation with 0.5 ppm of diquat, the decaying vegetation appeared to benefit certain benthic organisms, such as the Oligochaeta, as indicated by their increase in number (Tatum and Blackburn, 1962). There were indications, however, that this concentration of diquat acted as either direct or chronic poison to chironomids.

TABLE 64. The LC_{50} for various fish to diquat.

Formulation	Fish Species	Exposure (hr)	LC_{50} (ppm)	Source
Dibromide	Lake Emerald shiner	[1] 24	16.8	Swabey and Schenk, 1963
Salt	Largemouth bass	72	24	Surber and Pickering, 1962
Dibromide	Harlequin fish	[3] 24	78	Alabaster, 1969
"	Rainbow trout	[3] 24	90	"
Salt	Bluegill	96	91	Surber and Pickering, 1962
"	Fathead minnow	96	140	"
Dibromide	Lake Emerald shiner	[1] 96	180	Swabey and Schenk, 1963
Salt	Striped bass	96	210	Wellborn, 1969
"	Rainbow trout	48	12.3	FWPCA, 1968
"	Chinook salmon	48	28.5	Bond, Lewis and Fryer, 1960
"	Chinook salmon	48	28.5	Bohmont, 1967

[1] Medium hard water.
[2] Soft water.
[3] Tap water.

White shrimp exposed to 1.0 ppm of diquat for 48 hours showed no noticeable effects (Butler, 1965). The median immobilization concentration of diquat to *Daphnia magna* was 7.1 ppm (Crosby and Tucker, 1966).

Plants

Phytoplankton exposed to 1.0 ppm of diquat for 4 hours showed a 46-percent decrease in productivity (Butler, 1965).

Application of diquat at 0.5 ppm resulted in excellent control of an aquatic weed (*Lagarosiphon major*), but a massive growth of *Nitella* replaced *L. major* (Fish, 1966). The numbers of chironomid larvae also increased significantly.

Treatment of bean plants with 1,000 ppm of diquat reduced the ability of the fungus *Trichoderma viride* to compete with *Fusarium culmorum* (a pathogen that causes wilt disease in beans and other plants) (Wilkinson, 1969). The diquat, in reducing *T. viride*, allowed *F. culmorum* numbers to increase and infest the bean leaves, which resulted in plant damage.

Tatum and Blackburn (1962) reported that 0.5 ppm treatment with diquat in ponds adversely affected plankton, but the plankton recovered rapidly.

Biological Concentration

Esters of diquat accumulated in sunfish after exposure to sublethal concentrations fed in both laboratory and field tests (Cope, 1966b). When rainbow trout, however, were exposed to water containing 1 ppm of diquat (salt) for 80 days, only 0.09 ppm of diquat was found in the tissue, indicating that rainbow trout do not concentrate this diquat (salt) formulation (Cope, 1966).

Persistence

Diquat applied to ponds at a rate of 2.5 ppm persisted in the water for 7 to 27 days without a build-up in the hydrosoil (Grzenda, Nicholson and Cox, 1966).

DIURON

Mammals

The LD_{50} for the rat was 3,400 mg/kg to diuron when the mammal was fed the stated dosage orally (WSA, 1967).

Birds

The LD_{50} for young mallards was >5,000 mg/kg to diuron when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; for bobwhites, 2,000 to 3,200 ppm; and for coturnix, >5,000 ppm of diuron in diets of 2-week-old birds when fed treated food for 5 days followed by untreated food for 3 days (Heath et al., 1970).

Fishes

The 48-hour LC_{50} for largemouth bass and coho salmon to diuron was 42 ppm and 16 ppm, respectively (Bond, Lewis and Fryer, 1959). Bluegills were reported to be resistant, whereas white crappies were killed at concentrations as low as 6 ppm of diuron. Rainbow trout survived 60 ppm of diuron for 99 hours.

The 48-hour LC_{50} for rainbow trout exposed to diuron was 4,300 ppb (FWPCA, 1968).

The 24-hour LC_{50} for bluegills exposed to diuron at temperatures of 12.7°C, 18.3°C, and 23.8°C was 27,000 ppb, 17,000 ppb, and 9,700 ppb, respectively (Macek, Hutchinson and Cope, 1969).

The 96-hour LC_{50} for striped bass to diuron was 3.1 ppm (Wellborn, 1969).

Diuron was tested against various species of fish in different formulations, and the EC_{50} was as follows: bluegill, 5.7 ppm with emulsifiable diuron-TCA; brown bullhead, 11 ppm with diuron 80-percent wettable powder; and bluegill, 25 ppm with diuron 80-percent wettable powder (Walker, 1965). Clearly, the emulsifiable diuron-TCA formulation had far greater toxicity to bluegills.

The 24-hour LC_{50} of bluegills to diuron was 19 ppm (Cope, 1965a); however, Bohmont (1967) reported the 48-hour LC_{50} for diuron to bluegills as 74.0 ppm. Bohmont also reported the 48-hour LC_{50} for salmon as 16 ppm. Butler (1965) reported that in white mullet exposed to 6.3 ppm of diuron for 48 hours a 50-percent mortality resulted.

Small ponds were stocked with fingerling bluegills and treated with diuron (wettable powder) at 0.5, 1.5, and 3.0 ppm (McCraren, Cope and Eller, 1969). Oxygen level in the water decreased sharply in some of the ponds 9 days after treatment and remained low for 3 to 4 days more. In about 20 percent of the fish in the 3.0-ppm treatment, gill lamellae were ruptured and hemorrhagic. The only deaths among the fish were in cages in the 3.0-ppm treatment. The fish in all treatments grew more slowly than those in the control.

Molluscs

The exposure of eastern oysters to 1.8 ppm of diuron for 96 hours resulted in a 50-percent decrease in shell growth (Butler, 1965).

Arthropods

The LC_{50} for various arthropods to diuron is found in table 69.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to diuron was 2,000 ppb and 1,400 ppb, respectively (Sanders and Cope, 1966).

Brown shrimp exposed to 1.0 ppm of diuron for 48 hours showed no noticeable effect (Butler, 1965). The median immobilization concentration of diuron to *Daphnia magna* was 47 ppm (Crosby and Tucker, 1966).

About 50 hours after the treatment of the ponds with diuron at both 1.5 and 3.0 ppm, numerous distressed and dead midges, emerging mayflies, and dragonfly naiads were present on the water surface (McCraren, Cope and Eller, 1969). No invertebrates were observed on the surface of the untreated ponds.

Phytoplankton

Phytoplankton exposed to 1.0 ppm of diuron for 4 hours showed an 87-percent decrease in productivity (Butler, 1965).

Persistence

In moist-loam soil diuron applied at a rate of 1 to 3 lb/A persisted for 3 to 6 months with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

Diuron applied at 9 lb/A persisted in soil for >15 months (Weldon and Timmons, 1961).

Diuron residue persisted in the vegetation for some 90 days after treatment of ponds with dosages ranging from 0.5 to 3.0 ppm, and the residues in the mud persisted for 120 days (McCraren, Cope, and Eller, 1969).

TABLE 65. The LC_{50} for various arthropods to diuron

Arthropod Species	Exposure (hr)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcys californica*)	24	3.6	Sanders and Cope, 1968
Amphipod (*Gammarus lacustris*)	24	0.700	Sanders, 1969
" (*G. lacustris*)	48	0.380	FWPCA, 1968
Waterflea (*Daphnia pulex*)	48	1.4	"
Stonefly (*P. californica*)	48	2.8	"

DMPA

Mammals

The LD_{50} for female rats was 270 mg/kg; for guinea pigs, 210 mg/kg; and for dogs and cats, >1,000 mg/kg to DMPA when the animals were fed the stated dosages orally (WSA, 1967).

Birds

The LD_{50} for chickens was 1,000 mg/kg to DMPA when the birds were fed the stated dosage orally (WSA, 1967).

Arthropods

DMPA applied for control of crabgrass (8 to 16 oz/1000 sq. ft.) eliminated about 99 percent of the ant hills and indicated its insecticidal properties (Watson and Leasure, 1959).

DNBP

Mammals

Applications of DNBP for weed control in crops at rates of 1 to 6 lb/A were reported to have killed some rabbits in England, mainly through the ingestion of contaminated food (Edson, 1954 in Springer, 1957).

DNBP applied in a 0.25-percent solution to pastures strongly repelled the cattle from the treated vegetation (Grigsby and Farwell, 1950). Also, when DNBP (ammonium salt) was applied in a 0.12-percent solution to pastures, some repellent effect was measured against grazing cattle.

Birds

The lethal dose of DNBP to pheasants was 15 mg/kg (Paludan, 1953 in Springer, 1957).

Some pheasants and songbirds were poisoned when ingesting food from crop areas treated with 1 to 6 lb/A of DNBP (Edson, 1954 in Springer, 1957).

Fishes

Cope (1946 in Springer, 1957) reported that in laboratory tests small trout were killed with concentrations of 100 ppm of DNBP and 12 ppm of its ammonium salt.

Goldfish exposed for 24 hours to 0.1 ppm of DNBP showed no effect, but at 0.4 ppm there was 100-percent mortality (WSA, 1967).

The 24-hour LC_{50} for harlequin fish to DNBP was 0 ppm (Alabaster, 1969).

Arthropods

Cope (1946 in Springer, 1957) reported that some stoneflies and caddice flies survived when exposed to concentrations of 100 ppm of DNBP and 12 ppm of its ammonium salt.

Plants

The treatment of 9 weed species with a sublethal dosage of DNBP (0.03 lb/A) caused a decrease of 0 to 47-percent potassium nitrate content in 7 species (Frank and Grigsby, 1957). The nitrate content increased in 2 species—in *Eupatorium maculatum* from 8.9 mg to 23.7 mg (dry weight), a 165-percent increase.

Persistence

DNBP applied at 15 lb/A persisted in soil for 4 to ≥8 weeks (Warren, 1956).

ENDOTHALL

Mammals

The LD_{50} for the rat was 38 to 51 mg/kg to endothall acid when the mammal was fed the stated dosages orally (WSA, 1967).

Fishes

The LC_{50} for various fish to endothall is found in table 66.

Bluegills have tolerated endothall at 100 ppm for at least 21 days, when the test was discontinued (Lindaberry, 1961). In other tests by the investigator no mortality was observed in redfin, redsided shiner, and bluntnose minnow with endothall at 40 ppm; likewise, salmon, rainbow trout, and bass showed no mortality at 10 ppm. Based on these tests, the investigator concluded that normal use at 1 to 9 ppm should provide a wide margin of safety to fish.

Hiltibran (1967) reported that bluegills, green sunfish, lake chubsucker and smallmouth bass fry survived a concentration of 25 ppm of endothall for 8 days at the termination of the experiment.

Endothall applications (4 treatments at 1 ppm) appeared to have no effect upon the resident populations of bluegills and largemouth bass, but were effective in eliminating the submergent aquatic vegetation (Johnson, 1965).

An investigation of the persistence of endothall salt in fish revealed that 50 percent of the chemical was lost in ≤3 weeks (Walker, 1963).

Arthropods

The 24-hour LC_{50} for an amphipod (*Gammarus lacustris*) exposed to endothall (dipotassium salt) and endothall was ≥100 ppm and 9 ppm, respectively (Sanders, 1969).

Concentrations of greater than 1 ppm of endothall killed all bottom organisms in the ponds (Walker, 1962).

Nebeker and Gaufin (1964) reported that the LC_{50} for the amphipod crustacean *G. lacustris* to endothall was above 320 ppm.

The median immobilization concentration of endothall to *Daphnia magna* was found to be 46 ppm (Crosby and Tucker, 1966).

TABLE 66. The LC_{50} for various fish to endothall.

Formulation	Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Cocoamine salt	Lake Emerald shiner	[1] 24	0.15	Swabey and Schenk, 1963
Acid	Bluegill	72	428	Davis and Hughes, 1963
	Bluegill	[1] 24	450	Surber and Pickering, 1962
	Fathead minnow	[1] 24	>560	"
	Largemouth bass	[1] 24	>560	"
	Harlequin fish	24	565	Alabaster, 1969
	Bluegill	48	0.257	FWPCA, 1968
Copper	Rainbow trout	48	0.290	"
Dimethylamine	Rainbow trout	48	1.150	"
Acid	Salmon	48	136	Bohmont, 1967
	Redfin shiner	96	95	Walker, 1963
	Redsided shiner	96	105	"
	Bluntnose minnow	96	130	"
	Largemouth bass	96	130	"
	Bluegill	96	125	"
	Redear sunfish	96	135	"
	Carp	96	175	"
	Goldfish hybrid	96	175	"
	Yellow bullhead	97	175	"
	Black bullhead	96	180	"

[1] Medium hard water
[2] Soft water

Biological Concentration

Walker (1962) reported that at concentrations of 0.1 to 0.6 ppm, bottom organisms concentrated endothall approximately 200-fold in 3 weeks, but reported (1963) that he could not detect any absorption of the endothall compound in the fish after exposure to sublethal dosages in aquaria.

EPTC

Mammals

The LD_{50} for the rat was 1,630 mg/kg and for mice, 3,160 mg/kg to EPTC when the mammals were fed the stated dosages orally (USDI, 1970b).

Fishes

The exposure of white mullet to 20 ppm of EPTC for 48 hours caused a 10-percent mortality (Butler, 1963).

Molluscs

Eastern oysters exposed to 5.0 ppm of EPTC for 96 hours showed a 43-percent decrease in shell growth (Butler, 1963).

Arthropods

The exposure of white shrimp to 0.63 ppm of EPTC for 48 hours resulted in a 50-percent mortality or paralysis (Butler, 1963).

Phytoplankton

The exposure of phytoplankton to 1.0 ppm of EPTC for 4 hours caused no decrease in productivity (Butler, 1963).

Microorganisms

EPTC at normal dosages (2 to 6 lb/A in WSA, 1967) did not cause a reduction in the nitrification in soil, (Balicka and Sobieszczanski, 1969a in Balicka, 1969), but cellulose decomposition in the soil was impaired after its application (Sobieszczanski, 1969 in Balicka, 1969).

Persistence

In moist-loam soil EPTC applied at a rate of 2 to 6 lb/A persisted for 3 to 9 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

EPTC applied at 4 ppm in soil persisted for 8 months (Sheets, 1959).

FENAC

Mammals

The LD_{50} for the rat was 1,780 mg/kg when the mammal was fed the stated dosage orally (USDI, 1970b).

Birds

The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; and for coturnix, >5,000 ppm of fenac in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970).

Fishes

The LC_{50} for various fish to fenac is found in table 67.

Hiltibran (1967) reported that bluegill and lake chub-sucker fry survived a concentration of 20 ppm for 8 days, or until the experiment was terminated.

Arthropods

The LC_{50} for various arthropods to fenac is found in table 68.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to fenac (sodium salt) was 6,600 ppb and 4,500 ppb, respectively (Sanders and Cope, 1966).

TABLE 67. The LC_{50} for various fish to fenac.

Formulation	Fish Species	Exposure (hours)	LC_{50} (ppm)	Source
Sodium salt	Rainbow trout	24	10	Cope, 1965
"	Redear sunfish	24	12	"
"	Bluegill	24	50	Cope, 1965a
Acid	Bluegill	72	61	"
"	Rainbow trout	48	7.5	Bohmont, 1967
Sodium	Rainbow trout	48	7.5	FWPCA, 1968
Acid	Rainbow trout	48	16.5	"
"	Bluegill	48	19.0	Bohmont, 1967

TABLE 68. The LC_{50} for various arthropods to fenac.

Formulation	Arthropod Species	Exposure (hours)	LC_{50} (ppm)	Source
Sodium salt	Amphipod (*Gammarus lacustris*)	96	23	Sanders, 1969
"	Stonefly (*Pteronarcys californica*)	96	170	Sanders and Cope, 1968
"	" (*P. californica*)	96	320	"
"	Waterflea (*Daphnia pulex*)	48	4.5	FWPCA, 1968
"	Amphipod (*G. lacustris*)	48	18	"
Acid	Stonefly (*P. californica*)	48	70	"
Sodium salt	" (*P. californica*)	48	80	"

The median immobilization concentration of fenac (sodium salt) to *Daphnia magna* was > 100 ppm (Crosby and Tucker, 1966). The 24-hour LC_{50} for stonefly nymphs (*Pteronarcys*) to fenac (acid) and fenac (sodium salt) was 180 ppm and 270 ppm, respectively (Cope, 1965a).

Microorganisms

Fenac applied at the rate of 2 to 60 lb/A did not affect *Streptomyces* (Bounds and Colmer, 1964).

Persistence

Fenac applied to soil persisted for ≥ 18 months (Dowler, Sand and Robinson, 1963).

Fenac applied to a pond at 4 ppm persisted in the water for more than 202 days, with some of the herbicide also found in the hydrosoil (Grzenda, Nicholson and Cox, 1966).

FENURON

Mammals

The LD_{50} for the rat was 7,500 mg/kg to fenuron when the mammal was fed the stated dosage orally (USDI, 1970b).

Birds

Bobwhite quail fed fenuron at a maximum dosage of 2,500 mg/kg increased in weight faster than the birds in the control group (Bergstrand and Klimstra, 1962).

The LC_{50} for mallards was ≥5,000 ppm; for pheasants, ≥5,000 ppm; and for coturnix, ≥5,000 ppm of fenuron in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

Fishes

Spot were not affected by a 48-hour exposure to 1.0 ppm of fenuron (Butler, 1965).

Fenuron as a wettable powder had an EC_{50} for bluegills of about 55 ppm, whereas the emulsifiable fenuron-TCA was found to have an EC_{50} of about 6.8 ppm (Walker, 1965).

Hiltibran (1967) reported that bluegill, green sunfish, lake chub-sucker, and smallmouth bass fry survived a concentration of 10 ppm of fenuron-TCA for 8 days, or until the termination of the experiment.

Persistence

Fenuron applied to soil persisted at detectable levels for about 8 months (Kearney, Nash and Isensee, 1969).

FLUOMETURON

Mammals

The LD_{50} for female rats was 7,900 mg/kg; for female mice, 2,400 mg/kg; and for dogs, 10,000 mg/kg to fluometuron when the mammals were fed the stated dosages orally (WSA, 1967).

Birds

The LD_{50} for young mallards was $>2{,}000$ mg/kg to fluometuron when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The LC_{50} for killifish to fluometuron was >25 ppm, and a 100-percent mortality occurred with rainbow trout at >60 ppm (WSA, 1967). No exposure time was given.

Arthropods

All shrimp (*Gammarus pulex*) were killed at a dosage of 60 ppm (WSA, 1967). No exposure time was given.

IOXYNIL

Mammals

The LD_{50} for the rat was 110 mg/kg to ioxynil when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 24-hour LC_{50} for harlequin fish to ioxynil and ioxynil (sodium salt) was 0.28 ppm and 74 ppm, respectively (Alabaster, 1969).

LENACIL

Mammals

The LD_{50} for the rat was 11,000 mg/kg to lenacil when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 24-hour LC_{50} for harlequin fish to lenacil was about 50 ppm (Alabaster, 1969).

LFN

Fishes

The 48-hour LC_{50} for rainbow trout exposed to LFN was 79 ppb (FWPCA, 1968).

LINURON

Mammals

The LD_{50} for the rat was 4,000 mg/kg to linuron when the mammal was fed the stated dosage orally (USDI, 1970b).

Arthropods

An investigation by Edwards (1964) demonstrated that linuron at recommended application rates (0.5 to 3 lb/A in WSA, 1967) did not affect the numbers of soil animals.

Microorganisms

Linuron at normal concentrations in the bacterial medium had only temporary effects on the growth of three strains of *Bacillus* sp., but did not affect the growth of *Pseudomonas phaseoli* (Balicka and Krasel, 1969).

During 4 years of applying linuron at 5.4 lb/A, no change in the number of microorganisms in the soil was observed, employing various techniques for microorganism determination (Balicka and Sobieszczanski, 1969b in Balicka, 1969); linuron, however, caused apparent changes in the composition of microorganisms associated with roots or the treated plants (Balicka, 1969).

Linuron did not cause any change in nitrification of the soil (Balicka and Sobieszczanski, 1969a in Balicka, 1969), but cellulose decomposition in soil was impaired (Sobieszczanski, 1969 in Balicka, 1969). Treating soil with linuron caused an increase in the number of *Azotobacter* (Balicka, 1969).

Persistence

Linuron applied to soil persisted for about 4 months (Kearney, Nash and Isensee, 1969).

MALEIC HYDRAZIDE

Mammals

The LD_{50} for the rat was 4,000 mg/kg to maleic hydrazide when the mammal was fed the stated dosage orally (Spector, 1955).

Fishes

No mortality was observed with bluegills and fathead minnows when exposed to maleic hydrazide at 10 ppm (WSA, 1967).

Amphibians

Maleic hydrazide as a 1-percent solution killed 60 percent of the larvae of *Ambystoma punctatum* during a 10-day exposure (Greulach, McKenzie and Stacy, 1961).

Arthropods and Nematodes

With treatment of maleic hydrazide (0.08-percent solution) all *Daphnia* and *Cyclops* were killed (Greulach, McKenzie and Stacy, 1961).

Courtney, Peabody and Austenson (1962) reported that maleic hydrazide applied at a rate of 8 lb/A to colonial bentgrass reduced the number of bentgrass nematodes by 62 percent.

Plants

Fults and Payne (1956) found that treating bean, sugar-beet, and potato plants with maleic hydrazide (2,000 ppm) as a spray significantly increased the total free amino-acids in sugarbeet and potato, but not in the bean.

Sublethal dosages (1.0 lb/A) of maleic hydrazide applied to 9 species of weeds caused a decrease of 11 to 46 percent in the level of potassium nitrate in 6 species of the plants (the decline of 46 percent occurred in *Solanum dulcamara*) (Frank and Grigsby, 1957). There was a 48-percent increase in nitrate for *Impatiens biflora* and a 100-percent increase for *Eupatorium maculatum* (8.9 to 18.1 mg/g dry weight).

Persistence

In moist-loam soil maleic hydrazide applied at a rate of 3 to 6 lb/A persisted for 4 to 5 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

Maleic hydrazide applied at 5.0 ppm to soil persisted for ≥8 weeks (Hoffman, Parups and Carson, 1962).

MCPA

Mammals

The LD_{50} for the rat was 700 mg/kg to MCPA when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Birds

Chickens, exposed daily for 14 days to grass sprayed with MCPA (25-percent active agent) at a rate of ½ oz/gal of water and 2½ oz/gal, experienced a 60- to 97-percent reduction in egg production, respectively (Dobson, 1954). The fertility or hatchability of the eggs was unchanged and the chickens maintained their weight.

Fishes

Cope (1965) reported that the 96-hour LC_{50} of bluegills to MCPA was 10 ppm; but Davis and Hughes (1963) reported a 24-hour LC_{50} for MCPA (alkyl amine) as 168.5 ppm.

The exposure of longnose killifish to 75 ppm of MCPA amine for 48 hours resulted in a 50-percent mortality (Butler, 1963).

Molluscs

The exposure of oysters to 1.0 ppm of MCPA amine for 96 hours had no noticeable effect on shell growth (Butler, 1963).

Arthropods

Guilhon (1951) reported that honeybees succumbed rapidly to MCPA at levels of 5 to 8 μg/bee. The author also suspected that some mortality to bees may take place through the transport of MCPA-contaminated pollen to the hive. Jones and Connell (1954) calculated that the LD_{50} of honeybees to MCPA was 104.7 μg/bee. This figure is much higher than the 5- to 8-μg susceptibility given by Guilhon.

MCPA did not appear to have any appreciable effect on the total numbers of micro-arthropods in garden turf, even when the turf was treated at levels 10 times the normal dosage (Rapoport and Cangioli, 1963). Edwards (1964) also reported no noticeable effect from MCPA on soil animals. MCPA (sodium salt) at 2 lb/A applied for 8 years caused no changes in the number of Acari, Collembola, Insecta, and other Arthropoda (Davis, 1965).

The median immobilization concentration of MCPA to *Daphnia magna* was >100 ppm (Crosby and Tucker, 1966).

Plants

Swanson and Shaw (1954) demonstrated that the hydrocyanic acid content of Sudan grass increased by 58 percent (control, HCN 38 mg/100 g fresh wt. versus MCPA, 60 mg/100 g) in plots treated with 1 lb/A of MCPA.

When 9 species of weeds were treated with a sublethal dose (0.25 lb/A), the potassium nitrate level in 7 species decreased 8 to 80 percent (Frank and Grigsby, 1957). In 2 species, and especially *Impatiens biflora*, there was a 181-percent increase in potassium nitrate (9.7 to 26.0 mg/g dry weight) after this treatment.

The exposure of phytoplankton to 1.0 ppm of MCPA amine for 4 hours caused no noticeable decrease in productivity (Butler, 1963).

Microorganisms

Applications of MCPA at levels below about 8 lb/A did not inhibit the nodulation of legumes significantly (Elfadl and Fahmy, 1958). They also observed no significant injury to other soil microorganisms at dosages below 8 lb/A.

Persistence

In moist-loam soil MCPA applied at a rate of ½ to 9 lb/A persisted for 1 to 4 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

MCPA applied at 25 ppm in soil persisted for >100 days (Burger, MacRae and Alexander, 1962).

MCPB

Mammals

The LD_{50} for rats was 680 mg/kg to MCPB when the mammals were fed the stated dosage orally (FCH, 1970).

Arthropods

The use of MCPB at what was implied to be normal dosages reduced the number of arthropods by 50 percent and biomass by 66 percent in gray partridge habitats (Southwood, 1969). Arthropods play an important role in diets of partridge chicks.

Persistence

MCPB applied at 26 ppm to soil persisted for 54 days (Burger, MacRae and Alexander, 1962).

MERPHOS

Mammals

The LD_{50} for rats was 1,272 mg/kg to Merphos when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

When chickens were fed Merphos at a dosage of 800 mg/kg, the chickens developed leg weakness (Gaines, 1969).

MOLINATE

Mammals

The LD_{50} for male rats was 501 to 720 mg/kg, and for rabbits, >2,500 mg/kg to molinate when the mammals were fed the stated dosages orally (FCH, 1970).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to molinate was 290 ppb (FWPCA, 1968).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles exposed to molinate was 33 ppm (Sanders, 1970).

Arthropods

The 48-hour LC_{50} for stoneflies (*Pteronarcys californica*) exposed to molinate was 3,500 ppb (FWPCA, 1968).

The estimated 24-hour LC_{50} for stonefly nymphs (*P. californica*) to molinate was 3.3 ppm (Sanders and Cope, 1968).

The 24-hour LC_{50} for an amphipod (*Gammarus lacustris*) exposed to molinate was 5,800 ppb (Sanders, 1969).

MONOXONE

Mammals

The LD_{50} for the rat was 300 to 400 mg/kg to Monoxone when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for rainbow trout to Monoxone was 2,000 ppm (Alabaster, 1969).

MONURON

Mammals

The LD_{50} for the rat was 3,600 to 3,700 mg/kg to monuron when the mammal was fed the stated dosages orally (USDI, 1970b).

Birds

The LC_{50} for mallards was >5,000 ppm; for pheasants, 4,000 to 5,070 ppm; and for coturnix, >5,000 ppm of monuron in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

Fishes

Some mortality was observed in small golden shiners at a dosage of 20 ppm of monuron in ponds (Springer, 1957). The 24-hour LC_{50} for channel catfish was 75.0 ppm (Clemens and Sneed, 1959), and the 48-hour LC_{50} for mullet was 16.3 ppm (Butler, 1965). Monuron as an 80-percent wettable powder had an EC_{50} of 38 ppm, whereas an emulsifiable monuron was more toxic to bluegill (with an EC_{50} about 1.8 ppm) (Walker, 1962). The 48-hour LC_{50} for salmon to monuron was 110.0 ppm (Bohmont, 1967).

Hiltibran (1967) reported that bluegill, green sunfish, lake chub-sucker, and smallmouth bass fry survived a concentration of 10 ppm of monuron for 8, 5, 8, and 4 days, respectively. This experiment was terminated at 8 days.

Mollusca

The exposure of oysters to 2.0 ppm of monuron for 96 hours caused a 12-percent decrease in shell growth (Butler, 1965).

Arthropods and Annelids

At a dosage of 20 ppm of monuron applied to water, no mortality was observed in amphipods and isopods (Springer, 1957).

White shrimp exposed to 1.0 ppm of monuron for 48 hours exhibited no noticeable effects (Butler, 1965). The immobilization concentration of monuron to *Daphnia magna* was 100 ppm (Crosby and Tucker, 1966).

Earthworms were immersed in solutions of monuron for 2 hours, and at 1 ppm 10 percent were killed, whereas at 100 ppm 100 percent were killed (Martin and Wiggans, 1959).

Monuron applied at 10 lb/A significantly reduced the number of wireworms, millipedes, earthworms, springtails, and mites in soil 14 months after application (Fox, 1964). There was little or no effect 3 months after application.

Monuron at a dosage of 1 to 2 ppm reduced the average number of bottom-dwelling fish-food organisms 3 months after treatment (Walker, 1965). The weed-clinging species, such as dragonfly and damselfly, and the caddice fly and mayfly nymphs were significantly reduced.

Plants

Monuron applied to a lagoon at a rate of 60 lb/A killed many trees growing along the edge of the lagoon and the stream which drained the lagoon (Baumgartner, 1955). The trees, in order of susceptibility, were as follows: cottonwood, sycamore, box-elder, elm (*Ulmus* sp.), and ash (*Fraxinus* sp.).

The exposure of phytoplankton to 1.0 ppm of monuron for 4 hours caused a 94-percent mortality (Butler, 1965).

Although monuron was effective against algae at 1.5 ppm and higher plants at 5 ppm, as well as relatively non-toxic to fish, the chemical was reported as dangerous to many kinds of trees and shrubs whose roots were in contact with the treated water (Martin and Wiggans, 1959).

Microorganisms

The urea known as monuron was reported to be a powerful inhibitor of soil nitrification (Quastel and Scholefield, 1953). Otten, Dawson and Schreiber (1957) also reported that monuron at normal application rates (1 to 5 lb/A in WSA, 1967) inhibited soil nitrification. However, Hale, Hulcher and Chappell (1957) reported that monuron at 50 ppm had no noticeable effect upon nitrifying microorganisms.

Persistence

In moist-loam soil monuron applied at a rate of 1 to 3 lb/A persisted for 3 to 6 months with little or no leaching, under summertime conditions in a temperature climate (Klingman, 1961).

Monuron applied at 0.5 and 20 lb/A persisted in soil for 28 weeks (Holly and Roberts, 1963) and 3 years (Birk, 1955), respectively.

NAPHTHA

Fishes

The 48-hour LC_{50} for rainbow trout to naphtha was 5,600 ppb (FWPCA, 1968).

Arthropods

The LC_{50} for various arthropods to naphtha is found in table 60.

TABLE 60. The LC_{50} for various arthropods to naphtha.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	7	Sanders, 1969
Stonefly (*Pteronarcys californica*)	24	11	Sanders and Cope, 1968
Waterflea (*Daphnia pulex*)	48	3.7	FWPCA, 1968
Stonefly (*P. californica*)	48	5	"
Amphipod (*G. lacustris*)	48	3.6	"

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to naphtha was 7,600 ppb and 3,700 ppb, respectively (Sanders and Cope, 1966).

NEBURON

Mammals

The LD_{50} for the rat was >11,000 mg/kg to neburon when the mammal was fed the stated dosage orally (USDI, 1970b).

Fishes

Neburon appears to be the most toxic of the substituted-urea herbicides. For spot the 48-hour LC_{50} was 0.32 ppm (Butler, 1965). Walker (1965) reported the toxicities to various fish as follows: bluntnose minnow, 0.6 ppm; redear, 0.8 ppm; and the redfin shiner, 0.9 ppm.

Molluscs

The exposure of oysters to 0.41 ppm of neburon for 96 hours caused a 50-percent decrease in shell growth (Butler, 1965). Clam and snail populations doubled in number after treatment of ponds with neburon at 1 to 10 ppm (Walker, 1962).

Arthropods and Annelids

White shrimp exposed to 0.55 ppm of neburon for 48 hours showed a 50-percent mortality or paralysis (Butler, 1965).

A treatment of neburon ranging from 1 to 10 ppm reduced population of mayfly nymphs (Walker, 1962). In contrast, the following bottom organisms actually doubled in numbers after exposure to neburon: dragonfly nymphs, damselfly nymphs, and aquatic worms.

The number of bottom-dwelling fish-food organisms one year after treatment with neburon at 1 to 10 ppm was 1/10 that in untreated ponds (Walker, 1965). The most susceptible species were mayfly nymphs, common midges, and aquatic worms.

Phytoplankton

The exposure of phytoplankton to 1.0 ppm of neburon for 4 hours resulted in a 90-percent mortality (Butler, 1965).

Persistence

Neburon applied at 27 lb/A persisted in soil for 6 weeks (Quaglia, 1960).

In moist-loam soil neburon applied at a rate of 2 to 8 lb/A persisted for 3 to 6 months with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

PARAQUAT

Mammals

The LD_{50} for the rat was 150 mg/kg to paraquat when the mammal was fed the stated dosage orally (WSA, 1967).

Fishes

The exposure of longnose killifish to 1.0 ppm of paraquat for 48 hours had no noticeable effect (Butler, 1965).

When a pond was treated with 1 ppm of paraquat, no acute toxic effect was observed in rainbow trout, green sunfish, bluegills, or channel catfish (House et al., 1967).

The 24-hour LC_{50} for bluegills to paraquat was 400 ppm (Davis and Hughes, 1963).

The 24-hour LC_{50} for harlequin fish to paraquat and paraquat di(methyl)-chloride was 840 ppm and 45 ppm, respectively (Alabaster, 1969).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles and chorus frog tadpoles exposed to paraquat was 84 ppm and 43 ppm, respectively (Sanders, 1970).

Molluscs

The exposure of oysters to 1.0 ppm of paraquat for 96 hours had no noticeable effect on shell growth (Butler, 1965).

Arthropods and Annelids

House et al. (1967) reported that stoneflies were not affected by exposure to 1,000 ppm of paraquat for 96 hours.

The 48-hour LC_{50} for waterfleas *Daphnia pulex* and amphipods *Gammarus lacustris* exposed to paraquat was 3.7 ppm and 18 ppm, respectively (FWPCA, 1968).

Stonefly nymphs (*P. californica*) exposed to paraquat for 96 hours at 100 ppm were not affected (Sanders and Cope, 1968).

The 24-hour LC_{50} for an amphipod (*G. lacustris*) exposed to paraquat was 58 ppm (Sanders, 1969).

The exposure of brown shrimp to 1.0 ppm of paraquat for 48 hours had no noticeable effect (Butler, 1965).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *D. pulex*, to paraquat was 4 ppm and 3.7 ppm, respectively (Sanders and Cope, 1966).

The median immobilization concentration of paraquat to *Daphnia magna* was 11.0 ppm (Crosby and Tucker, 1966).

Mellanby (1967) reported that paraquat may be used to destroy all vegetation in a field ready for reseeding without plowing. He suggested that this process was less harmful to soil fauna than the usual cultivation. Earthworms, for example, which were frequently destroyed by cultivation survived the paraquat treatment.

Plants

The exposure of phytoplankton to 1.0 ppm of paraquat for 4 hours resulted in a 55-percent decrease in productivity (Butler, 1965).

Biological Concentration

In both laboratory and field tests paraquat accumulated in bluegills after their exposure to sublethal concentrations (Cope, 1966b).

Persistence

Paraquat applied to ponds at rates between 0.1 and 2.5 ppm persisted in the water for between 8 and 23 days; there was no buildup of the herbicide in the hydrosoil (Grzenda, Nicholson and Cox, 1966).

PCP

Mammals

The LD_{50} for the rat was 27 to 80 mg/kg to PCP when the mammal was fed the stated dosages orally (USDI, 1970b).

Vegetation treated with a 1-percent solution of PCP strongly repelled cattle (Grigsby and Farwell, 1950).

PCP, an herbicide, fungicide, and insecticide, was reported also to repel porcupines (Welch, 1954 in Springer, 1957).

Birds

The LD_{50} for pheasant was 4,000 to 5,000 mg/kg and for coturnix, 5,000 to 6,000 mg/kg to PCP when the birds were fed the toxicant in feed for 5 days plus 3 days of clean food (Heath et al., 1970).

Fishes

Ouchi fish seemed to discriminate PCP at levels of 0.2 and 0.3 ppm and avoided the treated areas (Tomiyama and Kawabe, 1962).

Molluscs

Molluscs, able to survive for 20 days in a concentration of 0.5 ppm (Tomiyama, Kobayashi and Kawabe, 1962), were more resistant to PCP than fish. A Japanese shellfish of commercial importance, *Venerupis philippinarum*, was sensitive to PCP at a dosage of 0.1 ppm.

Microorganisms

PCP in excess of 4 lb/A suppressed the activity of soil microorganisms (Kratochvil, 1950).

Persistence

PCP applied to soil (no dosage given) persisted for >5 years (Hetrick, 1952).

In moist-loam soil PCP applied at a rate of 5 to 20 lb/A persisted for 1 to 5 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

PICLORAM

Mammals

The LD_{50} for the rat was 8,200 mg/kg; for the mouse, 2,000 to 4,000 mg/kg; for the rabbit, about 2,000 mg/kg; and for the guinea pig, about 3,000 mg/kg to picloram when the mammals were fed the stated dosages orally (WSA, 1967).

Birds

The LD_{50} for chicks was 6,000 mg/kg (WSA, 1967); for young mallards, >2,000 mg/kg; and for young pheasants, >2,000 mg/kg (Tucker and Crabtree, 1970) to picloram when the birds were given the stated dosages orally in a capsule. The LC_{50} for mallards was >5,000 ppm and for pheasants, >5,000 ppm of picloram in diets of 2-week-old birds when fed treated feed for 5 days followed by 3 days of clean feed (Heath et al., 1972).

Fishes

The LC_{50} for various fish to picloram is found in table 70.

Picloram at 1 ppm with an exposure of 48 hours had no effect on guppies (Hardy, 1966).

Different species of fish have different tolerance levels to picloram, and this tolerance generally increases with increasing temperature (table 71). Rainbow trout were most tolerant to the triisopropanolamine salt (LC_{50}=67 ppm) and least tolerant to the isooctyl ester (LC_{50}=3.6 ppm).

Molluscs

Snails were found to survive picloram at a dosage of 380 ppm, but there was a 100-percent mortality at a dosage of 580 ppm (*Lynn*, 1965). Picloram at 1 ppm with an exposure of 48 hours did not affect the shell growth of eastern oysters (Butler, 1965).

Arthropods

The estimated 24-hour LC_{50} for stonefly nymphs (*Pteronarcys californica*) to picloram was 120 ppm (Sanders and Cope, 1968).

The 48-hour LC_{50} for amphipods (*Gammarus lacustris*) exposed to picloram was 48,000 ppb (FWPCA, 1968).

The 24-hour LC_{50} for an amphipod (*G. lacustris*) exposed to picloram was 50,000 ppb (Sanders, 1969).

Daphnia survived a 24-hour exposure to picloram at 380 ppm, but 95 percent were killed at a concentration of 580 ppm (Lynn, 1965).

Picloram at 1 ppm was reported to have no effect on brown shrimp when exposed for 48 hours (USDI, 1966a).

Hardy (1966) reported that picloram at 1 ppm did not affect the growth and reproduction of *Daphnia*, and there was no increase of concentration of this chemical in the tissue.

TABLE 70. The LC_{50} for various fish to picloram

Formulation	Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Acid	Fathead minnow	24	64	Weimer et al., 1967
Potassium salt	Harlequin fish	24	66	Alabaster, 1969
Acid	Fathead minnow	24	135	Lynn, 1965
"	Rainbow trout	24	180	Weimer et al., 1967
"	Green sunfish	24	180	"
"	Brown trout	24	200	"
"	Rainbow trout	24	230	Lynn, 1965
"	Brown trout	24	240	"
"	Brook trout	24	260	Weimer et al., 1967
"	Brook trout	24	420	Lynn, 1965
"	Green sunfish	24	420	"
"	Black bullhead	24	420	"
"	Rainbow trout	48	3.6	FWPCA, 1968

Table 71. The 24-hour LC_{50} for various fish to picloram formulations (Kenaga, 1969).

Formulation	Fish Species	LC_{50} (ppm)	Temp. (°F)
Acid	Bass	19.7	75
"	Bluegill	26.5	63
"	Goldfish	27-36	75
"	Coho salmon	29.0	53?
"	Rainbow trout	34	55
Triisopropanolamine salt	Rainbow trout	379.0	60
Triethylamine salt	Rainbow trout	43.4	"
"	Channel catfish	70.5	60
"	Goldfish	90.6	"
Potassium salt	Channel catfish	41	"
"	Bluegill	60	"
Isooctyl ester	Channel catfish	9.3	63
"	Rainbow trout	3.6	55
"	Rainbow trout	9.6	60
"	Channel catfish	16.4	60
"	Goldfish	27.0	"

Plants

Picloram and prometone at 27 lb/A were effective in preventing refoliation in tropical forests for more than 24 months (Dowler, Forestier and Tschirley, 1968).

Algae were unaffected at 1 ppm concentration of picloram in water (Hardy, 1966).

Microorganisms

Dosages up to 50 ppm of picloram did not reduce the growth of the fungus *Aspergillus niger* (Arnold, Santelmann and Lynd, 1966). *A. niger* was found to degrade picloram, but at a slower rate than it did 2,4-D.

The herbicide picloram at levels of 100 ppm had no measureable effect on populations of bacteria and fungi found in the soil and did not reduce nitrification (Goring et al., 1967).

Food Chain

In a food chain study algae, *Daphnia*, goldfish, and guppies were all reared together and exposed to a sublethal concentration of picloram (1 ppm). Over a 10-week period, there was no alteration in the normal growth of algae, *Daphnia*, or goldfish, and for a 6-month exposure there was no change in growth in the guppies (Lynn, 1965).

Persistence

Picloram applied at 5 lb/A persisted in soil for >500 days (Hamaker et al., 1968).

Picloram applied at a rate of 32 oz/A affected barley, alfalfa, and soybeans for a 9-month period after application (Herr, Stroube and Ray, 1966). Picloram persisted up to a year in soil (20 percent remaining) (Hamaker, Youngson and Goring, 1967).

PROMETONE

Mammals

The LD_{50} for the rat was 2,980 mg/kg to prometone when the mammal was fed the stated dosage orally (USDI, 1970b).

Fishes

The exposure of spot to 1.0 ppm of prometone for 48 hours had no noticeable effect (Butler, 1963).

Molluscs

The exposure of oysters to 1.0 ppm of prometone for 96 hours had no noticeable effect on shell growth (Butler, 1963).

Arthropods

The exposure of pink shrimp to 1.0 ppm of prometone for 48 hours had no noticeable effect (Butler, 1963).

Persistence

Prometone applied at 8 lb/A persisted in soil for >24 weeks (Holly and Roberts, 1963).

PROMETRYNE

Mammals

The LD$_{50}$ for the rat was 3,750 mg/kg to prometryne when the mammal was fed the stated dosage orally (USDI, 1970b).

Fishes

The spot exposed to 1.0 ppm of prometryne for 48 hours exhibited no noticeable effects (Butler, 1963).

Molluscs

The exposure of oysters to 1.0 ppm of prometryne for 96 hours caused a 10-percent decrease in shell growth (Butler, 1963).

Arthropods

Pink shrimp exposed to 1.0 ppm of prometryne for 48 hours were unaffected by the chemical (Butler, 1963).

Microorganisms

Some acceleration of nitrification was observed in soil treated with prometryne, but the total production of nitrates did not increase (Balicka and Sobieszczanski, 1969a, in Balicka, 1969). During 4 years of applying prometryne at 5.4 lb/A, no change in the number of microorganisms in the soil was found, no matter what medium was used for microorganism determination (Balicka and Sobieszczanski, 1969b in Balicka, 1969).

Persistence

Prometryne applied to soil persisted for about 3 months (Kearney, Nash and Isensee, 1969).

PROPAZINE

Mammals

The LD$_{50}$ for rats was 5,000 mg/kg to propazine when the mammals were fed the stated dosage orally (FCH, 1970).

Fishes

The 48-hour LC$_{50}$ for rainbow trout exposed to propazine was 7,800 ppb (FWPCA, 1968).

Persistence

Propazine applied at 0.5 lb/A persisted in soil for 11 to 24 weeks (Holly and Roberts, 1963).

PROPHAM

Mammals

The LD$_{50}$ for the rat was 5,000 mg/kg to propham when the mammal was fed the stated dosage orally (WSA, 1967).

Fishes

Propham caused no immediate danger or mortality to fish at a concentration of 10 ppm (Surber, 1948).

The 24-hour LC$_{50}$ for bluegills to propham was 32 ppm (Cope, 1965a).

Arthropods and Annelids

The 48-hour EC$_{50}$ (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to propham was 10 ppm and 10 ppm, respectively (Sanders and Cope, 1966).

The 24-hour LC$_{50}$ for an amphipod (*Gammarus lacustris*) exposed to propham was 20 ppm (Sanders, 1969).

Propham applied at about 8.55 lb/A caused a mortality of 13 percent in *Allolobophora caliginosa* and 42 percent in *Lumbricus castaneus* (earthworms) (Van der Drift, 1963).

Persistence

Propham applied at 4 ppm in soil persisted for 4 weeks (Burschel and Freed, 1959).

In moist-loam soil propham applied at a rate of 4 to 8 lb/A persisted for 2 to 4 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

PYRAZON

Mammals

The LD_{50} for the rat was 3,600 mg/kg to pyrazon when the mammal was fed the stated dosage orally (Neumeyer, Gibbons and Trask, 1969).

Fishes

The 24-hour LC_{50} for harlequin fish to pyrazon was 40 ppm (Alabaster, 1969).

REGULOX

Fishes

The 24-hour LC_{50} for rainbow trout to regulox was 85 ppm (Alabaster, 1969).

SESONE

Mammals

The LD_{50} for the rat was 730 mg/kg to Sesone when the mammal was fed the stated dosage orally (UCC, 1966).

Persistence

Sesone applied at 2.1 lb/A persisted in soil for 6 weeks (Quaglia, 1960).

SILVEX

Mammals

The LD_{50} for the rat was 1,070 mg/kg; for the mouse, 2,140 mg/kg; for the rabbit, 850 mg/kg; and for the guinea pig, 850 mg/kg to silvex when the mammals were fed the stated dosages orally (Mullison, 1966 in House et al., 1967).

Birds

When mallard ducks were fed silvex at daily dosages of 2,500 and 5,000 ppm, reproduction was suppressed nearly 100 percent (USDI, 1970a). The LC_{50} for pheasants was 5,000 to 5,000 ppm, and for coturnix, >5,000 ppm of silvex (acid) in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days; and an LC_{50} for coturnix was >5,000 ppm of silvex (butoxyethanol ester) (Heath et al., 1970).

Fishes

The LC_{50} for various fish to silvex is found in table 79.

The variability in the toxicities reported with silvex and bluegills may be due to the formulation employed, as shown by the data in table 79. The ester formulation were generally more toxic, because they more effectively penetrate the body of the fish.

Butler (1963) reported further evidence that the ester formulations of silvex were more toxic to fish; he found the 48-hour LC_{50} for spot to silvex to be 0.36 ppm.

TABLE 79. The LC_{50} for various fish to silvex.

Formulation	Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Acid	Bluegills	18	70	Cope, 1965
"	Bluegills	¹24	2.9	Surber and Pickering, 1962
"	Fathead minnow	¹24	8.0	"
"	Bluegills	24	18	Cope, 1965a
"	Rainbow trout	24	97	"
"	Harlequin fish	24	48	Alabaster, 1969
"	Bluegills	48	0.80	Bohmont, 1967
PGBEE²	Rainbow trout	48	0.650	FWPCA, 1968
BEE³	Bluegills	48	1.2	"
Acid	Salmon	48	1.06	Bohmont, 1967
Isooctyl	Bluegills	48	1.4	FWPCA, 1968

[1] Soft water.
[2] Propylene glycol butyl ether ester.
[3] Butoxyethanol ester.

TABLE 73. The LC_{50} of bluegills to silvex formulations (Hughes and Davis, 1963).

Silvex Chemical	24 hr	48 hr
Potassium salt	83	83
Isooctyl ether [1]	15.5	14.1
Isooctyl ester [1]	3.7	3.7
Isooctyl ester [1]	1.4	1.4
Propylene glycol butyl ether ester	16.6	16.6
Butoxyethanol ester	1.2	1.2

[1] Different batches of the same formulation.

Swabey and Schenk (1963) reported that the 24-hour LC_{50} for Lake Emerald shiner in medium-hard water to granular silvex (potassium salt) was 840 ppm; to liquid silvex (potassium salt), 490 ppm; and to liquid silvex (butyl ester), 4.0 ppm. The ester formulation was again quite toxic to fish.

Hiltibran (1967) reported that bluegill, lake chub-sucker, and smallmouth bass fry survived a concentration of 20 ppm of liquid silvex (potassium salt) for 8 days or the duration of the experiment; green sunfish fry survived 10 ppm of granular silvex (potassium salt) also for 8 days. Mullison (1966) provides a good summary of the influence of silvex on fish populations; in general, the safe dosage of silvex appears to be somewhat below 3 ppm. At dosages of 3 ppm of silvex and above, he reported liver degeneration, testicular degenerative lesions, and abnormal spermatozoa.

The effects of silvex on resident game fish populations in ponds were measured when treatments were made for the eradication of submergent aquatic vegetation. Silvex at 2 ppm with 3 applications had no effect on largemouth bass in one pond, or on brook trout in 2 ponds; and silvex with one application at 3 ppm in one pond had no effect on rainbow or brook trout (Johnson, 1965).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles and chorus frog tadpoles exposed to silvex was 22 ppm and 20 ppm, respectively (Sanders, 1970).

Molluscs

The exposure of oysters to 1.0 ppm of silvex (ester) for 96 hours caused a 23-percent decrease in shell growth (Butler, 1963).

Arthropods and Annelids

The exposure of brown shrimp to 0.94 ppm of silvex (ester) for 48 hours resulted in a 60-percent mortality or paralysis (Butler, 1963).

The 24-hour LC_{50} for stonefly nymphs (*Pteronarcys*) to silvex was 5.6 ppm (Cope, 1965a).

The median immobilization concentration of silvex (sodium salt) to *Daphnia magna* was 100 ppm (Crosby and Tucker, 1966).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to silvex was 2,400 ppb and 2,000 ppb, respectively (Sanders and Cope, 1966).

The 48-hour LC_{50} for waterflea (*D. pulex*) exposed to silvex (propylene glycol butyl ether ester) was 2,000 ppb (FWPCA, 1968).

The estimated 24-hour LC_{50} for stonefly nymphs (*P. californica*) to silvex was 5.2 ppm (Sanders and Cope, 1968).

Stonefly nymphs tolerated only 0.32 ppm of silvex for 96 hours. *Daphnia* appeared to be more resistant than the stoneflies to this chemical and tolerated the normal treatment dosage of 2 ppm (House et al., 1967).

The bottom fauna in portions of plastic-enclosed farm ponds were investigated before and after treatment with silvex at dosages ranging from 2.8 ppm to 4.6 ppm (Harp and Campbell, 1964). The standing crop of some bottom fauna doubled numerically in the treated areas, compared with the untreated. The increase in numbers of chironomids and oligochaetes in the treated areas was attributed to the increase in organic matter resulting from the decay of poisoned aquatic plants. The dipteran (*Chaoborus*) increased greatly in the treated areas, and populations of dragonfly nymphs (*Libellula*) increased one year after the treatment. Snails, leeches, and damselflies were apparently unaffected by the treatment of silvex. The dipteran (*Chrysops*) decreased rapidly in the treated pond and reappeared only during the last 2 months of the study and only in the enclosures receiving the lower concentration of silvex. The authors considered the impact of the sudden treatment with herbicide and the resulting decaying plant material in a pond community quite similar to that of the effect of organic sewage pollution on an aquatic community.

Plants

The exposure of phytoplankton to 1.0 ppm of silvex (ester) for 4 hours caused a 76-percent decrease in productivity (Butler, 1963).

Silvex at 2 ppm had no adverse effect on either phytoplankton or zooplankton, which were at the base of the food chain in small test ponds (Cowell, 1965).

Microorganisms

Silvex did not affect *Streptomyces* at 1 to 80 lb/A (Bounds and Colmer, 1964).

Biological Concentration

Esters of silvex accumulated in bluegills after exposure to sublethal concentrations in both laboratory and field tests (Cope, 1966b). No dosages were given.

Persistence

In moist-loam soil silvex applied at a rate of ½ to 3 lb/A persisted for 3 to 5 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

Silvex applied at 20 ppm to soil persisted for >103 days (Burger, MacRae and Alexander, 1962).

SIMAZINE

Mammals

The LD_{50} for the rat was >5,000 mg/kg to simazine when the mammal was fed the stated dosage orally (House et al., 1967).

Birds

The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; and for coturnix, >5,000 ppm of simazine in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

Fishes

The LC_{50} for various fish to simazine is found in table 74.

An investigation of the persistence of simazine in fish revealed that 50 percent of the chemical was lost in <3 days (Mauck, 1969).

Molluscs, Arthropods, and Annelids

The 48-hour LC_{50} for stoneflies (*Pteronarcys californica*) and amphipods (*Gammarus lacustris*) exposed to simazine was 50 ppm and 21 ppm, respectively (FWPCA, 1968).

The 24-hour LC_{50} for an amphipod (*G. lacustris*) exposed to simazine was 50 ppm (Sanders, 1969).

TABLE 74. The LC_{50} for various fish to simazine.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Striped bass	96	0.60	Wellborn, 1969
Rainbow trout	24	85	Cope, 1965a
Bluegills	24	130	"
Rainbow trout	48	5	FWPCA, 1968
Rainbow trout	48	56	Bohmont, 1967
Bluegills	48	118	"

Arthropods appear to be susceptible to simazine. The following species of bottom-dwelling organisms were reduced by 50 percent or more after an application of simazine ranging from 0.5 to 10 ppm: mayflies, mosquitoes, biting midges, damselfly nymphs, water beetles, aquatic worms, leeches, and snails (Walker, 1962). According to Walker (1964), a dosage of about 20 ppm killed about 50 percent of the common midges and aquatic worms.

DeVries (1962) found that only 1 of 8 *Lumbricus* and none of 32 *Helodrilus* were killed at both 8 and 12 lb/A of simazine after 32 days' exposure in potted soil.

Edwards (1964) investigated the influence of simazine at normal dosages (2 to 4 lb/A in WSA, 1967) on soil animals and reported a reduction in the numbers of animals in the treated soil by one-third to one-half. Predatory mites, hemiedaphic Collembola, and particularly the Isotomidae, were most affected by simazine. Earthworms, enchytraeid worms, dipterous and coleopterous larvae, and populations of other mites and springtails also increased, and significant differences between the numbers of those in treated and untreated soil were still obvious 3 to 4 months after the chemical had been applied.

Microorganisms

The normal application rates (2 to 4 lb/A in WSA, 1967) of simazine for weed control did not significantly affect the relative numbers of fungi and bacteria in soil or the growth of some fungi (Eno, 1962). Farmer, Benoit and Chappell (1965) noted inhibition of nitrification with simazine at concentrations of 6 ppm or greater. Other investigators observed some acceleration of nitrification in soil treated with normal application rates of simazine, but reported no increase in the total production of nitrates (Balicka and Sobieszczanski, 1969a in Balicka, 1969). The acceleration of nitrification might be due to larger dosages used. Simazine-treated soil also resulted in an increase in the number of *Azotobacter* (Balicka, 1963).

Persistence

In moist-loam soil simazine applied at a rate of 1 to 4 lb/A persisted for 3 to 6 months with little or no leaching under summertime conditions in a temperate climate (Klingman, 1961).

Simazine applied at 2 lb/A persisted in soil for 17 months (Talbert and Fletchall, 1964).

SODIUM ARSENITE

Mammals

The LD_{50} for the rat was 10 to 50 mg/kg (USDA, 1967 in Hume et al., 1967) and for the mouse, 31 mg/kg (Meliere, 1959) to sodium arsenite when the mammals were fed the stated dosages orally.

Birds

Mallard ducks tolerated 8 mg/day of sodium arsenite for a period in which the total dose reached 973 mg/kg in the ducks (USDI, 1963).

Fishes

The LC_{50} for various fish to sodium arsenite is found in table 75.

Bond (1960) reported sodium arsenite to be safe at dosages of 2 to 4 ppm arsenic trioxide in soft waters and 5 or 6 ppm in hard waters.

Rainbow trout (LD_{50} = 60 mg/kg) and bluegills (LD_{50} = 44 mg/kg) were relatively tolerant of sodium arsenite, compared with other herbicides (Crosby and Tucker, 1966).

Cope (1966) reported that a dosage of 4 ppm of sodium arsenite caused kidney and liver damage in bluegills.

Sodium arsenite applied at 5 ppm for the eradication of submergent aquatic vegetation in ponds had no effect on rainbow or brook trout populations (Johnson, 1965).

TABLE 75. The LC_{50} for various fish to sodium arsenite.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Lake Emerald shiner	[1] 24	13.5	Swabey and Schenk, 1963
Spottail minnow	24	29	Boschetti and McLoughlin, 1957
Bluegills	24	44	Cope, 1966a
Rainbow trout	24	100	″
Rainbow trout	48	36.5	FWPCA, 1968

[1] Medium hard water.

An investigation of the persistence of sodium arsenite in fish revealed that 80 percent of the chemical was lost in >16 weeks (Mauck, 1969).

Mollusca and Arthropoda

The minimum lethal dosages (ppm) of sodium arsenite producing a kill of fish-food organisms exceeding 95 percent are the following: *Daphnia*, 3.0; *Eucypris*, 6.0; *Hyalella*, 2.5; *Culex*, *Aedes*, and *Anopheles*, 6.0; and *Chironomus*, 10.0 (Zischkale, 1952). A concentration of 6.5 ppm of sodium arsenite immobilized 50 percent of *Daphnia magna*.

The estimated 24-hour LC_{50} for stonefly nymphs (*Pteronarcys californica*) to sodium arsenite was 140 ppm (Sanders and Cope, 1968).

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and

Daphnia pulex, to sodium arsenite was 1,400 ppb and 1,800 ppb, respectively (Sanders and Cope, 1966).

The 48-hour LC_{50} for waterfleas (*S. serrulatus*) exposed to sodium arsenite was 1,400 ppb (FWPCA, 1968).

Bond (1960) reported that fish and fish-food organisms may be harmed indirectly through the use of herbicides. When large amounts of vegetation are killed, the rapid decomposition may deplete oxygen, resulting in heavy kills of both fish and fish-food organisms.

Johnson (1965) reported that 3 to 8 ppm of sodium arsenite killed filamentous algae and submerged aquatic plants in ponds, but had no effect on the numbers of pond invertebrates such as chironomid larvae, beetle larvae and adults (Haliplidae), true bugs (Notonectidae and Dytiscidae), mayfly nymphs, damselfly nymphs, dragonfly nymphs, and amphipods. Walker (1962) however, reported that treating ponds with sodium arsenite at dosages from 2.5 to 20 ppm caused a 50-percent reduction in phantom midges, water bugs, and snails.

The 24-hour LC_{50} for stonefly nymphs (*Pteronarcys*) to sodium arsenite was 100 ppm (Cope, 1965a).

Phytoplankton and Zooplankton

Sodium arsenite at 4 ppm did not affect the number of phytoplankton, but did cause drastic reductions in the number of zooplankton (Cowell, 1965).

Biological Concentration

Cope (1966) reported that bluegill concentrated sodium arsenite in a few days from a level of 0.69 ppm in the water to 11.6 ppm in adult bluegills.

SODIUM CHLORATE

Mammals

The LD_{50} for the rat was about 5,000 mg/kg to sodium chlorate when the mammal was fed the stated dosage orally (WSA, 1967).

Birds

Sublethal dosages of sodium chlorate may have significant effects upon chickens, as indicated by a study of Dobson (1954) in which he exposed chickens daily for 14 days to grass sprayed daily with sodium chlorate at ½ lb/gal and 2 lb/gal of water. The low sodium chlorate treatment led to a 60-percent reduction in egg yield and the higher dosage, to a 90-percent reduction plus a decrease in fertility and hatchability. The exposed chickens also lost weight.

Fishes

The 24-hour LC_{50} for channel catfish to sodium chlorate was 5,157 ppm (Clemens and Sneed, 1959).

The 24-hour LC_{50} for harlequin fish to sodium chlorate was 8,000 ppm (Alabaster, 1969). The 24-hour LC_{50} for rainbow trout to Chlorax (sodium chlorate and sodium metaborate) and to sodium chlorate was 2,000 ppm and 4,200 ppm, respectively.

Persistence

Sodium chlorate applied at 800 lb/A persisted in soil for >1 year (Nelson, 1944).

SODIUM PENTACHLOROPHENATE

Mammals

The LD_{50} for the rat was 210 mg/kg to sodium pentachlorophenate when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for rainbow trout to sodium pentachlorophenate was 0.26 ppm (Alabaster, 1969).

The 24-hour LC_{50} to sodium pentachlorophenate for various fish was as follows: guchi fish at 0.60 ppm; warasube at 3.4 ppm; and eel at 0.20 ppm (Tomiyama and Kawabe, 1962).

Sodium pentachlorophenate applied to a creek at the rate of 9.5 ppm was reported to kill all catfish (*Ictalurus*), guppies, and eels (Springer, 1957).

Arthropods

Crayfish exposed to 9.5 ppm of sodium pentachlorophenate in creek water were unharmed (Springer, 1957).

2,4,5-T

Mammals

The LD_{50} for the rat was 500 mg/kg and for the dog, 100 mg/kg to 2,4,5-T when the mammals were fed the stated dosages orally (Spector, 1955).

Rowe and Hymas (1954) presented data indicating that the acute oral LD_{50} of 2,4,5-T to various species of mammals was about 500 mg/kg.

Birds

The LC_{50} for mallards was >5,000 ppm; for pheasants, 1,250 to 2,500 ppm; and for coturnix, >5,000 ppm of 2,4,5-T in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

The use of 2,4,5-T and 2,4-D for brush control under power lines improved the environment for ruffed grouse, as measured by an increase in grouse numbers (Bramble and Byrnes, 1958). The grouse were found on the edges within 150 to 200 feet of the right-of-way, rather than on the right-of-way itself. This emphasized the importance of the right-of-way as a creator of edge effects. Wild turkeys also made effective use of the right-of-way treated areas. The young turkeys were attracted to the cleared area for feeding on various insects, which were more abundant on the grassy right-of-way than within the wooded areas.

Sublethal concentrations of 2,4,5-T may have significant effects upon biological activities in birds. Chickens were exposed for 14 days to grass sprayed daily with 2,4,5-T (15-percent active agent) at ½ oz/gal of water and 2½ oz/gal (Dobson, 1954). The lower dosage led to a 9-percent reduction in egg yield, and the higher dosage to an 18-percent reduction, but there was no change in the fertility or hatchability of the eggs. The exposed chickens also lost some weight.

Fishes

See table 76 for the LC_{50} for various fish to 2,4,5-T.

When young silver salmon were exposed to a combination of 2,4,5-T and 2,4-D (about 10 percent of each chemical in the formulation) at concentrations of 50 ppm or more they were "immediately distressed and would snap their jaws, dart about the aquarium, and leap out of the water before loss of equilibrium and death" (Holland et al., 1960).

Mullet exposed to 50 ppm of 2,4,5-T for 48 hours exhibited no noticeable effects (Butler, 1965).

The 24-hour LC_{50} of bluegills to various 2,4,5-T formulations are presented in table 77 (Hughes and Davis, 1963). The ester formulations appeared to be most toxic to the fish probably due to more effective penetration. No attempt was made by Hughes and Davis to explain the wide

TABLE 76. The LC_{50} for various fish to 2,4,5-T

Formulation	Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Butyl ester	Harlequin fish	24	1.0	Alabaster, 1969
Isopropyl ester	Bluegill	24	1.6	Davis and Hughes, 1963
Oleic-1,3-propylene diamine	Bluegill	24	0.9	"
Acid	Rainbow trout	24	12	Alabaster, 1969
Triethylamine	Bluegill	24	55.7	Davis and Hughes, 1963
Acid	Bluegill	48	0.50	Bohmont, 1967
Propylene glycol butyl ether ester	Bluegill	48	[illegible]	FWPCA, 1968
Acid	Rainbow trout	48	1.3	Bohmont, 1967
Isopropyl ester	Bluegill	48	1.7	FWPCA, 1968
Isooctyl ester	Bluegill	48	16.7	"

variation in results obtained from the different batch lots of the same formulation.

Hiltibran (1967) reported that bluegill and green sunfish fry survived a concentration of 10 ppm for 8 days or the termination of the experiment.

Table 77. The 24-hour LC_{50} of bluegills to 2,4,5-T formulations (Hughes and Davis, 1963).

2,4,5-T	LC_{50} (ppm)
Dimethylamine	144
Isooctyl ester [1]	31
Isooctyl ester [1]	28
Isooctyl ester [1]	10.4
Propylene glycol butyl ethyl ester	17
Butoxyethanol ester	1.4

[1] Different batches of same formulation.

Molluscs

The exposure of oysters to 2.0 ppm of 2,4,5-T acid for 96 hours had no effect on shell growth (Butler, 1963).

Arthropods

The minimum lethal dosages (ppm) which produced a kill exceeding 25 percent with 2,4,5-T are listed for the following fish-food organisms: *Daphnia*, 1.5; *Eucypris*, 0.5; *Hyallela*, 0.7; *Palaemonetes*, 1.0; *Amphiagrion*, 7.5; *Pachydiplax* and *Tramea*, 8.0; and *Chironomus*, 6.0 (Zischkale, 1952).

The exposure of brown shrimp to 1.0 ppm of 2,4,5-T for 48 hours had no deleterious effects (Butler, 1963).

Plants

Fifteen days after black-cherry brush had been treated until wet with a 2,4,5-T concentration of 2,000 ppm, Grigsby and Ball (1952) reported that the hydrocyanic acid (HCN) content was reduced 88 percent (control=91.9 mg/100 g fresh wt; 2,4,5-T=10.8 mg/100 g).

Swanson and Shaw (1954) demonstrated that the hydrocyanic acid content of Sudan grass was increased by 80 percent (control, HCN 33 mg/100 g fresh wt. versus 2,4,5-T, 61 mg/100 g) in plots treated with 1 lb/A of 2,4,5-T.

When 9 species of weeds were treated with sublethal dosages (0.25 lb/A) of 2,4,5-T, the nitrate content of the plants decreased from 5 to 52 percent in 4 species and increased from 5 to 56 percent in 5 other species (Frank and Grigsby, 1957). The 56-percent increase (control, 6.8 mg/g dry wt. versus 2,4,5-T, 10.6 mg/g) in potassium nitrate occurred in *Impatiens biflora*.

The exposure of phytoplankton to 1.0 ppm of 2,4,5-T for 4 hours caused no decrease in productivity (Butler, 1963).

Microorganisms

Magee and Colmer (1955) reported that 2,4,5-T at 1,500 to 2,000 ppm produced an inhibition of respiration in *Azotobacter* sp. Bounds and Colmer (1964), however, found that 2,4,5-T did not affect *Streptomyces* at 2 and 50 lb/A.

Persistence

2,4,5-T applied at 5 ppm to soil persisted for 160 to >190 days (DeRose and Newman, 1947).

In moist-loam soil 2,4,5-T applied at a rate of ½ to 3 lb/A persisted for 2 to 5 weeks with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961). Sheets and Harris (1965), however, reported that 2,4,5-T generally persisted for about 5 months under moist soil conditions.

TAR DISTILLATE

Birds

Tar distillate had a significant effect when chickens were exposed daily for 14 days to grass sprayed daily with tar distillate winter wash (80 percent cresol and phenol as the active agents) at ½ pt and 2 pt made up to 1 gal of water (Dobson, 1954). The low tar-distillate treatment led to a 17-percent reduction in egg yield, and the higher dosage led to a 40-percent reduction, but there was no reduction in egg fertility or hatchability. The chickens did not lose weight after the exposure.

2,3,6-TBA

Mammals

The LD_{50} for the rat was 750 mg/kg to 2,3,6-TBA when the mammal was fed the stated dosage orally (PCOC, 1966).

Persistence

2,3,6-TBA applied at 1 to 8 lb/A persisted in soil for >18 months (Dowler, Sand and Robinson, 1963).

TCA

Mammals

The LD_{50} for the rat was 5,000 mg/kg; for the mouse, 3,640 mg/kg; and for the rabbit, 4,000 mg/kg to TCA when the mammals were fed the stated dosages orally (WSA, 1967).

Birds

The LD_{50} for chicks was 4,000 mg/kg to TCA when the chicks were fed the stated dosage orally (WSA, 1967).

Fishes

The 24-hour LC_{50} of channel catfish to TCA (90 percent) was 2,000 ppm (Clemens and Sneed, 1959). Bond, Lewis and Fryer (1960) also found that chinook salmon would survive a 48-hour exposure to 970 ppm of TCA. The results indicate that catfish and salmon were relatively tolerant of TCA.

TCA has been employed in aquatic habitats in combination with monuron, fenuron, and diuron. Combinations always resulted in increased toxicity to fish (Walker, 1965).

Arthropods and Annelids

TCA at a dosage of 60 lb/A was found to increase the number of millipedes, springtails, and mites in the soil, while decreasing the number of earthworms 14 months after treatment (Fox, 1964).

Microorganisms

TCA at normal application rates markedly suppressed the activity of microorganisms (Kratochvil, 1950). Otten, Dawson and Schreiber (1957) also reported that TCA at normal application rates reduced soil nitrification based on laboratory tests.

Persistence

TCA applied at 15 lb/A persisted in soil for 42 to 84 days (Rai and Hemmer, 1958).

In moist-loam soil TCA applied at a rate of 40 to 100 lb/A was found to persist for 50 to 90 days with little or no leaching, under summertime conditions in a temperate climate (Klingman, 1961).

TRIFLURALIN

Mammals

The LD_{50} for the rat was >10,000 mg/kg; for the mouse, 5,000 mg/kg; for the rabbit, >2,000 mg/kg; and for the dog, >2,000 mg/kg to trifluralin when the mammals were fed the stated dosages orally (WSA, 1967).

The LD_{50} for rats was >10,000 mg/kg to trifluralin when the mammals were fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for chickens was >2,000 mg/kg (WSA, 1967); for young mallards, >2,000 mg/kg; and for young pheasants, >2,000 mg/kg (Tucker and Crabtree, 1970) to trifluralin when the birds were given the stated dosages orally in a capsule.

Fishes

The 24-hour LC_{50} for bluegills and rainbow trout to trifluralin was 0.19 ppm and 0.21 ppm, respectively (Cope, 1965a).

The 24-hour LC_{50} for rainbow trout exposed to trifluralin at temperatures of 1.6°C, 7.2°C, and

12.7°C was 8.6 ppb, 230 ppb, and 98 ppb, respectively (Macek, Hutchinson and Cope, 1969); and the 24-hour LC_{50} for bluegills exposed at temperatures of 12.7°C, 18.3°C, and 23.8°C was 540 ppb, 560 ppb, and 190 ppb, respectively. As both temperature and time of exposure increased, the LC_{50} decreased for bluegills exposed to trifluralin (table 78).

TABLE 78. The effects of time and temperature on the toxicity of trifluralin to bluegills averaging 38 mm in length and 0.80 g in weight (Cope, 1965a).

Temperature, °F	LC_{50} (ppb)		
	24 hrs	48 hrs	96 hrs
85	10	8.4	8.4
75	190	66	47
65	300	200	135
55	530	390	210
45	1,300	590	280

The 48-hour LC_{50} for rainbow trout exposed to trifluralin was 11 ppb (FWPCA, 1968).

The 24-hour LC_{50} for harlequin fish to trifluralin was 0.5 ppm (Alabaster, 1969).

Amphibians

The 24-hour LC_{50} for Fowler's toad tadpoles exposed to trifluralin was 0.10 ppm (Sanders, 1970).

Arthropods

The LC_{50} for various arthropods to trifluralin is found in table 79.

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to trifluralin was 450 ppb and 240 ppb, respectively (Sanders and Cope, 1966).

Persistence

Trifluralin applied to soil persisted for about 6 months (Kearney, Nash and Isensee, 1969).

TABLE 79. The LC_{50} for various arthropods to trifluralin.

Arthropod Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Amphipod (*Gammarus lacustris*)	24	8.8	Sanders, 1969
Stonefly (*Pteronarcys californica*)	24	13	Sanders and Cope, 1968
" (*Pteronarcys* sp.)	24	13.0	Cope, 1965a
Waterflea (*Daphnia pulex*)	48	0.240	FWPCA, 1968
Stonefly (*P. californica*)	48	4.2	"
Amphipod (*G. lacustris*)	48	5.6	"

TRIOXONE

Fishes

The 24-hour LC_{50} for rainbow trout to trioxone was 12 ppm (Alabaster, 1969).

UREABOR

Fishes

The 24-hour LC_{50} for rainbow trout to ureabor was 975 ppm (Alabaster, 1969).

VERNOLATE

Mammals

The LD_{50} for the rat was 1,800 mg/kg to vernolate when the mammal was fed the stated dosage orally (WSA, 1967).

Fishes

The 24-hour LC_{50} to vernolate for bluegills was 9.7 ppm (Cope, 1965a) and for rainbow trout, 6.9 ppm (WSA, 1967). The 96-hour LC_{50} for three-spined stickleback to vernolate was 1 to 10 ppm (WSA, 1967).

The 48-hour LC_{50} for rainbow trout exposed to vernolate was 5,000 ppb (FWPCA, 1968).

Molluscs

The 96-hour EC_{50} (shell growth inhibition) for oysters to vernolate was greater than 1 ppm (maximum level tested) (WSA, 1967).

Arthropods

The 48-hour EC_{50} (loss of equilibrium or death) for brown shrimp to vernolate was greater than 1 ppm (maximum level tested) (WSA, 1967).

The 48-hour LC_{50} for amphipods (*Gammarus Lacustris*) exposed to vernolate was 25,000 ppb (FWPCA, 1968).

The 24-hour LC_{50} for an amphipod (*G. lacustris*) exposed to vernolate was 8,400 ppb (Sanders, 1969).

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PART IV

Fungicides

AMMONIUM CHLORIDE

Arthropoda

Ammonium chloride at 91 ppm in Lake Erie water was found to immobilize *Daphnia magna* within 64 hours (Anderson, 1948).

AMMONIUM HYDROXIDE

Amphibians

Ammonium hydroxide at 16 ppm was reported to be toxic to *Rana pipiens* and bullfrogs when exposed for 48 hours (Alabama, 1955).

BENZENETHIOL

Fishes

The exposure of brown trout, bluegill, and goldfish to 5 ppm of benzenethiol for 24 hours resulted in no mortality in any of the species (Spector, 1955).

BENZOIC ACID

Amphibians

The lethal dose of benzoic acid to frogs by subcutaneous injection was 100 to 200 mg/kg (Spector, 1955).

BIUREA

Fishes

The exposure of brown trout, bluegill, and goldfish to 6 ppm of biurea, a medical fungicide, for 24 hours resulted in no mortality in any of the species (Spector, 1955).

BUSAN

Fishes

The 24-hour LC$_{50}$ for harlequin fish to busan 90 and 881 was 1.8 ppm and 1.1 ppm, respectively (Alabaster, 1969).

CADMIUM SUCCINATE

Birds

The LC_{50} for pheasants was 1,350 to 1,400 ppm; for bobwhites, 1,700 to 1,800 ppm; and for coturnix, 2,000 to 2,200 ppm of cadmium succinate in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

CAFFEINE

Amphibians

The lethal dose of caffeine, a protectant material, to frogs by subcutaneous injection was 120 to 150 mg/kg in 4 to 5 days (Spector, 1955).

CAPTAFOL

Mammals

The LD_{50} for [illegible] 6,700 mg/kg to captafol when the [illegible] fed the stated dosage orally (FCH, 1970).

Birds

Captafol caused a 6.7-percent incidence of teratogenesis in chick embryos when the chemical was injected into eggs at dosages ranging from 5 to 20 ppm (Verrett et al., 1969). The incidence of abnormalities in control chick embryos was <2.0 percent. Most of the malformations occurred in the wings and legs.

Fishes

The 48-hour LC_{50} for channel catfish exposed to captafol was 31 ppb (FWPCA, 1968).

The 24-hour LC_{50} for harlequin fish to captafol was 0.032 ppm (Alabaster, 1969).

Arthropods

See table 80 for the LC_{50} for various arthropods to captafol.

TABLE 80. The LC_{50} for various arthropods to captafol.

Arthropod Species	Exposure (hours)	LC_{50} (ppm)	Source
Stonefly (*Pteronarcys californica*)	24	0.40	Sanders and Cope, 1968
Amphipod (*Gammarus lacustris*)	24	2.2	Sanders, 1969
Stonefly (*P. californica*)	48	0.150	FWPCA, 1968
Amphipod (*G. lacustris*)	48	6.5	"

CAPTAN

Mammals

The LD_{50} for the rat was 9,000 mg/kg to captan when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; for bobwhites, 2,000 to 4,000 ppm; and for coturnix, >5,000 ppm of captan in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

Captan caused a 7.8-percent incidence of teratogenesis in chick embryos when the chemical was injected into eggs at dosages ranging from 5 to 20 ppm (Verrett et al., 1969). The incidence of abnormalities in control chick embryos was <2.0 percent. Most of the malformations occurred in the legs and wings.

Arthropods and Annelids

Beran and Neururer (1955) reported that the LD_{50} of captan to honeybees was 2.44 µg per bee when fed orally.

Treatment of orchards at recommended rates (1 to 10 lb/A in Thomson, 1967) with captan caused little or no reduction in the numbers of beneficial predaceous and parasitic arthropods (MacPhee and Sanford, 1961). Schneider (1958) reported that captan at normal rates of application in orchards had no effect upon the parasitic wasp

Aphelinus mali. In the laboratory employing a normal spray concentration of 0.125 percent captan against the parasitic wasp *Mormoniella vitripennis* caused no mortality (Ankersmit et al., 1962).

Captan applied to potted apple trees at a dosage of 0.20 percent exhibited little or no toxicity to beneficial predatory mites (Van de Vrie, 1962). However, captain sprayed in orchards at a rate of 1 lb/100 gal of water caused some mortality of beneficial parasitic wasps (especially *Metaphycus helvolus*), but caused little or no mortality to beneficial predatory coccinellid beetles (Bartlett, 1963).

Captan applied to orchards at normal spray dosages did not harm beneficial predatory mites (*Typhlodromus* sp.) or such beneficial wasp parasites as *Mormoniella* sp. and *Aphelinus mali* (Bezemer, 1964).

Captan applied to apple trees at a concentration of 0.15 percent was reported by Van de Vrie (1967) to be harmless to the predatory bug *Anthocoris nemorum*, but to cause some mortality to the predatory bug *Orius* sp. and to the parasite *Aphelinus mali.* These results with *A. mali* do not agree with the findings of Schneider (1958), who reported no effect with captan.

Ulrich (1968) reported that captan remaining on a surface after treatment with a concentration of 1,000 ppm in water had little or no effect upon *Trichogramma* female adults (wasp parasite, primarily of Lepidoptera eggs) when exposed for 10 hours.

Earthworms, *Eisenia foetida*, were immersed for 2 hours in solutions containing captan (Martin and Wiggans, 1959). There was little mortality at exposure of 10 ppm, but at 100 ppm there was 100-percent mortality.

Soil treated with 15, 60, and 500 lb/A of captan failed to prove toxic to *Lumbricus* after 52 days' exposure (DeVries, 1962). *Helodrilus* mortality was 47 percent after 52 days' exposure to the 500 lb/A dose.

Persistence

Captan in soil persisted for >65 days (Munnecke, 1958).

When captan was well distributed in the soil, the fungicide had a half life of 1 to 2 days (Griffith and Matthews, 1969). However, when the material was applied in heavy concentrations on the surface of beads (simulating seeds), captan persisted; there was little change in concentration even after 21 days.

CARBOLIC ACID

Fishes

The 24-hour LC_{50} for channel catfish to carbolic acid was 16.7 ppm (Clemens and Sneed, 1959).

The 24-hour LC_{50} for harlequin fish to carbolic acid was 9.2 ppm (Alabaster, 1969).

The exposure of brown trout, bluegill, and goldfish to 5 ppm of carbolic acid (o-phenyl phenol) for 22 hours resulted in no mortality in any of the species (Spector, 1955).

Carbolic acid at 500 to 600 ppm was found to be deadly to fish in tests run in Russia (Demyanenko, 1931).

Amphibians

The lethal dose of carbolic acid (o-chlorophenol) to frogs by subcutaneous injection was 400 mg/kg (Spector, 1955).

CHLORANIL

Mammals

The LD_{50} for the rat was 4,000 mg/kg to chloranil when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

Neff and Meanley (1958 in Springer, 1957) reported that blackbirds were not repelled by chloranil (tetrachloro-p-benzoquinone). Pheasants were observed to eat seed-corn with chloranil with little or no harm (Landy and Cole, 1950).

Persistence

Chloranil applied to soil persisted for >20 days (Domsch, 1958).

CHLORINE

Amphibians

Chlorine at 0.25 ppm was toxic to *Hyla cinerea* and small *Rana pipiens* tadpoles within 12 hours at 70°F, and 2.0 ppm was required to kill large bullfrog tadpoles (Alabama, 1955).

CHLORONITROPROPANE

Mammals

The LD_{50} for rats was 107 mg/kg to chloronitropropane when the mammals were fed the stated dosage orally (*FCH*, 1970).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to chloronitropropane was 100 ppb (*FWPCA*, 1968).

Arthropods

The 48-hour LC_{50} for stoneflies (*Pteronarcys californica*) exposed to chloronitropropane was 5,800 ppb (*FWPCA*, 1968).

The 24-hour LC_{50} for an amphipod (*Gammarus lacustris*) exposed to chloronitropropane was 2,800 ppb (Sanders, 1969).

Microorganisms

Chloronitropropane at 10 ppm was found to stop soil activity of nitrifying microorganisms (Cawley and Broadbent, 1968).

COPPER CARBONATE

Mammals

Copper carbonate applied as a spray or painted on bark proved to be an effective repellent on plants for various species of rabbits, especially the white-tailed jackrabbit (Garlough, Welch and Spencer, 1942).

Birds

Copper carbonate applied to seed corn did not prevent pheasants from consuming the seed (Dambach and Leedy, 1949 in Springer, 1957). No report was made whether the fungicide was toxic to the pheasants.

COPPER OXYCHLORIDE

Fishes

The 48-hour LC_{50} for bluegill exposed to copper oxychloride was 1,100 ppb (*FWPCA*, 1968).

Arthropods

Schneider (1958) reported copper oxychloride applied to orchards at normal rates of application (2 to 5 lb/A in Thomson, 1967) to have no effect on populations of the beneficial parasitic wasp *Aphelinus mali*.

Van de Vrie (1962) reported that copper oxychloride applied to apple leaves at a concentration of 0.25 percent killed 84 percent of the predatory mites (*Typhlodromus tiliae* and *T. tiliarum*) after 7 days of exposure in the laboratory.

COPPER-8-QUINOLINOLATE

Fishes

The 24-hour LC_{50} for rainbow trout to copper-8-quinolinolate was 0.80 ppm (Alabaster, 1969).

COPPER SULFATE

Mammals

Hayne (1949) reported that 1 oz copper sulfate and 1.5 oz hydrated lime (Bordeaux mixture) in 1 gal of water applied to beans and cabbages repelled cottontail rabbits. The effectiveness was lost in a few days.

Bordeaux mixture at normal application rates (10 lb copper + 10 lb lime in 100 gal of water in

Thomson, 1967) to garden plants was reportedly effective in repelling rabbits and other rodents in gardens (Hildreth and Brown, 1955).

Copper sulfate and similar fungicides at normal application rates to crops have been found to poison sheep and chickens on farms (Antoine, 1966). The poisoning comes about through an accumulation of copper in the animals. For example, the daily intake of 25 mg during several months by sheep resulted in serious jaundice in these animals.

Birds

The LD_{50} for young mallards was >2,000 mg/kg and for pheasants, >2,000 mg/kg to Bordeaux mixture when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The 24-hour LC_{50} for striped bass to copper sulfate was 1.5 ppm (Wellborn, 1969).

The 48-hour LC_{50} for bluegill exposed to copper sulfate was 150 ppb (FWPCA, 1968).

The toxicity of copper sulfate to fish varies with the species and with the physical and chemical characteristics of the water. The chemical was especially toxic to trout in soft water (Bond, 1960). The highest concentrations of copper sulfate tolerated by various fish are shown in table 81. Trout were obviously the most sensitive, agreeing with Bond's results.

The margin of safety for fish in using copper sulfate for aquatic weed control was small (DeVaney, 1968). Dosages for weed control range from 0.08 ppm to 10.0 ppm for the control of various weeds (USDA, 1954).

Copper sulfate was found to be less toxic to fish in hard water than soft water (table 82).

Copper sulfate applied to 4 lakes in Minnesota at a rate of 0.12 to 0.50 ppm did not affect fish yields for the preceding 24 years, compared with 5 untreated lakes (Moyle, 1949).

Arthropods and Other Invertebrates

Stultz (1955) observed that all principal parasites were at relatively high densities after the use of copper fungicides in orchards at normal application rates. Bordeaux mixture (3 lb copper sulfate + 10 lb lime) at 2 lb/100 gal of water applied to orchards did not cause any harmful effects to beneficial arthropod predators and parasites (MacPhee and Sanford, 1961). Bartlett (1963) reported also that Bordeaux mixture at a high dosage of 40 lb/100 gal of water applied to orchards caused little or no mortality to beneficial parasitic wasps and predatory coccinellid beetles.

TABLE 81. The highest concentrations of copper sulfate tolerated by various fish (McKee and Wolf, 1963)

Fish	Dosage (ppm)	Fish	Dosage (ppm)
Trout	0.14	Perch	0.67
Carp	0.33	Largemouth bass and bluegill	0.80
Suckers	0.33	Sunfish	1.35
Catfish	0.40	Smallmouth bass	2.00
Pickerel	0.40		
Goldfish	0.50		

TABLE 82. Toxicity of copper sulfate (48-hr LC_{50}) to bluegills in water from 4 sources (McKee and Wolf, 1963)

LC_{50} (ppm)	Total Hardness (ppm)	Total Alkalinity (ppm)
0.8	15.0	16.7
8.0	86.0	166.0
10.0	100.0	245.0
45.0	183.0	1044.0

Copper sulfate applied at a rate of 0.05 to 0.08 ppm in ponds for control of algae resulted in an increase in copepods, cladocerans, rotifers, chaoborid larvae, and ostracods and other zooplankton (Crance, 1963).

Copper-containing fungicides which have been used extensively in orchards eliminated most animal life in the soil, including the large earthworm (*Lumbricus terrestris*) (Mellanby, 1967). Raw (1962) earlier reported that copper fungicides can almost eradicate earthworms from soil to which they are applied.

Plants

In some Minnesota lakes which had been treated for 26 years with copper sulfate, the blue-green algae *Aphanizomenon* appeared to have evolved increased resistance to copper sulfate (Moyle, 1949).

CORROSIVE SUBLIMATE

Mammals

The LD_{50} for the rat was 1 to 5 mg/kg to corrosive sublimate when the mammal was fed the stated dosages orally (FCH, 1970).

Fishes

Sticklebacks were able to survive for 10 days when exposed to corrosive sublimate at 8 ppb (Jones, 1939).

Arthropods

The toxicity limitation of corrosive sublimate with the crustacean *Daphnia* was less than 0.006 ppm (Anderson, 1948).

CRESOL

Fishes

The 96-hour LC_{50} for channel catfish to o-cresol was 66.8 ppm (Clemens and Sneed, 1959).

Cresol at 500 to 600 ppm was found to be deadly to fish in test runs in Russia (Demyanenko, 1951).

Amphibians

Both m-cresol and p-cresol were found to be lethal to frogs by subcutaneous injection with dosages of 350 mg/kg and 150 mg/kg, respectively (Spector, 1955). This fungicide is employed as a protectant chemical for materials such as wood.

CYANO(METHYLMERCURI)GUANIDINE

Birds

The LD_{50} for young mallards was 586 mg/kg; for young pheasants, 566 mg/kg; and for house sparrows, 500 to 900 mg/kg to cyano(methylmercuri)guanidine when the birds were given the stated dosages orally in a capsule (at 6.8 percent active ingredient) (Tucker and Crabtree, 1970).

CYCLOHEXAMIDE

Mammals

The LD_{50} for the rat was 2.5 mg/kg to cyclohexamide when the mammal was fed the stated dosage orally (FCH, 1970).

Cyclohexamide has been shown to be highly repellent to laboratory rats (Traub et al., 1950). At 1 ppm in water rats would die, rather than accept the treated water. The antibiotic was reported, however, to have limited repellency to four species of mice (Welch, 1954).

Birds

The LD_{50} for mallard ducks was 50 to 100 mg/kg to cyclohexamide when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Plants

Lemin and Thomas (1961) reported that cyclohexamide was taken up from a stem application and distributed to the upper stem and needles in eastern white pine seedlings, but was not translocated to the roots.

CYCLOPENTADIENE

Fishes

The exposure of brown trout and bluegill to 5 ppm of cyclopentadiene for 24 hours resulted in no mortality in either of the 2 species (Spector, 1956).

DELRAD

Fishes

The 48-hour LD_{50} for channel catfish to Delrad was 0.74 ppm (Clemens and Sneed, 1959).

Mollusca

Delrad at 0.5 ppm caused about 60 percent mortality of clam larvae, and 1 ppm caused 100 percent (Davis, 1961).

Arthropods

Delrad was found to be toxic to small crustaceans (copepods) at 1 ppm (Alabama, 1955, and Springer, 1957).

DEXON

Mammals

The LD_{50} for the rat was about 60 mg/kg to Dexon when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 48-hour LC_{50} for bluegill exposed to Dexon was 23,000 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for stoneflies (*Pteronarcys californica*) and amphipods (*Gammarus lacustris*) exposed to Dexon was 4,200 ppb and 6,800 ppb, respectively (FWPCA, 1968).

Microorganisms

Dexon at 100 and 200 mg/kg soil treatments decreased the number of detectable *Pythium* propagules and also decreased the root rot index when peas were planted in the treated soils (Alconero and Hagedorn, 1968). The number of *Fusarium* spp., *Actinomucor elegans*, and *Trichoderma* spp. appeared not to be affected by the chemical treatments.

Persistence

Soil treated with Dexon at a rate of 200 mg/kg still had residues of Dexon at 71 ppm one year later (Alconero and Hagedorn, 1968).

DICHLOFLUANID

Mammals

The LD_{50} for the male rat was 1,000 mg/kg to dichlofluanid when the mammal was fed the stated dosage orally (FCH, 1970).

Arthropods

At least 80 percent of a population of *Trichogramma* adult females, a beneficial wasp parasite of Lepidoptera eggs, when exposed to the mildew fungicide, dichlofluanid, remaining on a surface after treatment at a concentration of 750 ppm in water were killed within 16 hours (Ulrich, 1968).

DICHLONE

Mammals

The LD_{50} for the rat was 1,300 mg/kg to dichlone when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for young mallards was >2,000 mg/kg to dichlone when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Dichlone-treated rice seed was found not to have any repellent action and to be readily eaten by blackbirds (Neff and Meanley, 1956 in Springer, 1957).

Fishes

See table 60 for the LC_{50} for various fish to dichlone.

Dichlone was toxic to bluegills at 0.15 ppm and to fathead minnows at 0.25 ppm (no exposure time given) (Alabama, 1955).

Dichlone at a concentration of 0.1 ppm was reported to be toxic to fingerling largemouth bass in 22 hours and at 1 ppm, toxic to goldfish and bluegills in 8 hours (Alabama, 1954). However, Fitzgerald and Skoog (1954) reported that concentrations of 30 and 55 ppb of dichlone had no observed effect on fish and zooplankton when applied to a lake for control of blue-green algae.

Dichlone has been used for algae control at about 0.5 ppm; however, at concentrations of 0.05 ppm largemouth bass have been killed in aquarium tests in soft water (Bond, 1960).

TABLE 89. The LC_{50} for various fish to dichlone.

Fish Species	Exposure Time (hr)	LC_{50} (ppm)	Source
Rainbow trout	24	0.04	Alabaster, 1956
Channel catfish	96	0.14	Clemens and Sneed, 1959
Salmon	48	0.043	Bohmont, 1967
Rainbow trout	48	0.068	FWPCA, 1968

The 24-hour LC_{50} for an amphipod (*Gammarus lacustris*) exposed to dichlone was 5,000 ppb (Sanders, 1969).

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) and amphipods (*G. lacustris*) exposed to dichlone was 26 ppb and 11,500 ppb, respectively (FWPCA, 1968).

Dichlone applied to orchards in Nova Scotia at recommended application rates significantly reduced the populations of 30 to 40 percent of the beneficial parasitic and predaceous species in the treated crop area (MacPhee and Sanford, 1954 and 1956).

Stultz (1955) reported dichlone at normal application rates in orchards to reduce seriously the numbers of predaceous insects attacking the bud moth, especially the predator *Haplothrips faurei*. In contradiction to Stultz's results, MacPhee and Sanford (1961) reported that treatments of dichlone at recommended dosages (0.5 lb/100 gal of water) in orchards caused little or no reduction in the numbers of most beneficial predaceous and parasitic arthropods; however, dichlone did cause some reduction in the numbers of a mite predator (*Typhlodromus pyri*).

The median immobilization concentration of dichlone to *Daphnia magna* was 0.014 ppm (Crosby and Tucker, 1966).

DICHLOROPHEN

Mammals

The LD_{50} for the guinea pig was 1,250 mg/kg to dichlorophen when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for rainbow trout and harlequin fish to dichlorophen (sodium salt) was 0.32 ppm and 0.24 ppm, respectively (Alabaster, 1969).

DICLORAN

Mammals

The LD_{50} for the rat was >10,000 mg/kg to dicloran when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for mallards was >2,000 mg/kg to dicloran when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Plants

Lemin (1965) showed dicloran to be translocated by plants (tomato seedlings).

Microorganisms

Dicloran was found to inhibit progressively soil nitrification by microorganisms starting at 10 ppm, with complete inhibition occurring at 1,000 ppm (Caseley and Broadbent, 1968).

DINOCAP

Mammals

The LD_{50} for the rat was 980 mg/kg to dinocap when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to dinocap was 0.14 ppm (Alabaster, 1969).

Arthropods

Dinocap at a concentration of 0.25 percent (normal spray concentration: 0.025 percent) caused only a 9-percent mortality to the wasp parasite *Mormoniella vitripennis* (Ankersmit et al., 1962). Besemer (1964) supported these findings, reporting that dinocap applied at recommended dosages in orchards proved to be relatively harmless to *Mormoniella*, but slightly toxic to another wasp parasite, *Aphelinus mali*.

The wasp parasite *Trichogramma* was also found to be susceptible to dinocap (Ulrich, 1968). At least 65 percent of adult females of this wasp were killed after a 4-hour exposure to dinocap remaining on a surface after treatment at a concentration of 280 ppm in water. In the field Besemer (1964) reported that a single application of dinocap destroyed a portion of the *Trichogramma* population, important in the control of leafrollers in orchards.

Dinocap at a concentration of 0.06 percent was reported to kill 100 percent of the predatory mite populations (*Typhlodromus tiliae* and *T. tiliarum*) in laboratory tests (Van de Vrie, 1962). The field use of dinocap at recommended dosages in orchards was reported by Besemer (1964) to cause high mortalities to predacious mites.

Van de Vrie (1967) reported that dinocap applied to apple trees at a concentration of 0.06 percent caused no harm to the predatory bug *Anthocoris nemorum*, some reduction in the predatory bug *Orius* sp., and a high mortality to the parasite *Aphelinus mali*.

DITHIANON

Mammals

The LD_{50} for the rat was 1,015 mg/kg to dithianon when the mammal was fed the stated dosage orally (FCH, 1970).

Arthropods

Ulrich (1968) reported that dithianon remaining on a surface after treatment with a concentration of 600 ppm in water had little or no effect upon the wasp egg parasite *Trichogramma* (female adults) exposed for 10 hours.

DMTT

Mammals

The LD_{50} for the rat was 500 mg/kg to DMTT when the mammal was fed the stated dosage orally (FCH, 1970).

Persistence

DMTT applied to soil persisted for 4 days (Domsch, 1958).

DODINE

Mammals

The LD_{50} for the male rat was about 1,000 mg/kg to dodine when the mammal was fed the stated dosage orally (FCH, 1970).

Arthropods

Dodine applied to orchards at 0.75 lb/100 gal of water caused a reduction in the numbers of two mirid predators (*Deraeocoris nebulosus* and *Hyaliodes harti*) (MacPhee and Sanford, 1961).

Dodine remaining on a surface after treatment with a concentration of 500 ppm in water had little or no effect upon the egg parasite *Trichogramma* (adult females) when exposed for 10 hours (Ulrich, 1968).

DYRENE

Mammals

The LD_{50} for the rat was about 2,710 mg/kg to Dyrene when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for young mallards was >2,000 mg/kg to Dyrene when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Fishes

The 48-hour LC_{50} for bluegill exposed to Dyrene was 15 ppb (FWPCA, 1968).

Arthropods

The 48-hour LC_{50} for waterfleas (*Daphnia magna*) exposed to Dyrene was 490 ppb (FWPCA, 1968).

Phytoplankton

A 91.5-percent decrease in productivity of natural phytoplankton communities occurred when they were exposed for 4 hours to a concentration of 1.0 ppm of Dyrene (Butler, 1963).

EC–90

Fishes

The 24-hour LC_{50} for harlequin fish to EC-90 was 2.2 ppm (Alabaster, 1969).

FENTIN ACETATE

Mammals

The LD_{50} for the rat was 125 mg/kg to fentin acetate when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to fentin acetate was 0.08 ppm (Alabaster, 1969).

FENTIN HYDROXIDE

Mammals

The LD_{50} for the rat was 108 mg/kg to fentin hydroxide when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 48-hour LC_{50} for bluegill exposed to fentin hydroxide was 55 ppb (FWPCA, 1968).

FERBAM

Mammals

The LD_{50} for the rat was >17,000 mg/kg to ferbam when the mammal was fed the stated dosage orally (FCH, 1970).

In tests conducted by Hildreth and Brown (1955) ferbam was found not to be repellent to rabbits.

Fishes

The 96-hour LC_{50} for channel catfish to ferbam was 16.6 ppm (Clemens and Sneed, 1959), and Butler (1963) reported that the 24-hour LC_{50} for juvenile longnose killifish to ferbam was 1.0 ppm.

Ferbam applied to ponds at 0.5 ppm was reported by Eipper (1959) to cause blindness in one northern pike, a largemouth bass, and 2 common bluegills about 23 days after treatment. In addition, in aquarium tests ferbam was observed to cause severe fin erosion in all lots of 3-inch brook trout. Eipper and Forney (1954, in Springer, 1957) reported that ferbam killed fingerling brook trout at concentrations of 1 to 2 ppm, but did not appear to be lethal to pike, bass, or bluegill in ponds treated with 0.5 to 4 ppm.

Molluscs

A concentration of 0.075 ppm of ferbam in sea water caused a 50-percent decrease in eastern oyster shell growth during a 96-hour exposure (Butler, 1963).

Arthropods

Ferbam applied to Nova Scotia orchards at recommended dosages was found to reduce significantly about 22 percent of the numbers of species of beneficial predators and parasites in this crop ecosystem (MacPhee and Sanford, 1954). Further documentation of these results came from the investigations of Stultz (1955) in Nova Scotia. He reported that the wasp parasites *Meteorus trachynotus* and *Ascogaster quadridentata* were only rarely seen after several years' use of ferbam at normal application rates in orchards; however, he reported little or no change in the effectiveness of the parasite *Agathis laticinctus*. MacPhee and Sanford (1956), in later investigations of the influence of spray programs on the fauna of apple orchards, reported that ferbam at normal application rates caused significant reduction in the numbers of about 10 percent of the beneficial parasitic and predaceous species.

The parasitization of pest insect eggs by *Trichogramma* was significantly reduced (by about 75 percent) when ferbam was applied to orchards at recommended dosages in Germany (Stein, 1961).

In contrast with the above findings, Bartlett (1963) reported that ferbam at 1.75 lb/100 gal of water applied to orchards in California caused little or no mortality to beneficial parasitic wasps and predatory coccinellid beetles.

Plants

The effect of ferbam added to the soil annually at 200 lb/A from 1949 to 1953 was measured by growing various crop plants in the contaminated soil for several years following the treatments (MacPhee, Chisholm and MacEachern, 1960). With high residues of ferbam in the soil at time of growth, yields of the crop plants were as follows: beans, no effect; turnips, increased 1.7 times; carrots, no effect; tomatoes, little effect; and peas, no effect.

Ferbam applied at 0.5 ppm to 2 ponds killed 90 percent of the filamentous algae, but only 60 to 80 percent of *Cladophora* were killed in 2 ponds treated with 1.5 and 3 ppm, respectively (Eipper, 1959).

A 97.0-percent decrease in productivity of natural phytoplankton communities occurred when they were exposed for 4 hours to a concentration of 1.0 ppm of ferbam (Butler, 1963).

Persistence

Ferbam applied to soil persisted for 28 days (Jacques, Robinson and Chase, 1959).

FOLPET

Mammals

The LD_{50} for the rat was >10,000 mg/kg to folpet when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for young mallards was >2,000 mg/kg to folpet when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Folpet caused an 8.2-percent incidence of teratogenesis in chick embryos when the chemical was injected into eggs at dosages ranging from 5 to 20 ppm (Verrett et al., 1969). The incidence of abnormalities in control chick embryos was <2.0 percent. Most of the malformations occurred in the wings and legs.

Fishes

Butler (1963) reported that the 24-hour LC_{50} for juvenile white mullet and longnose killifish to folpet was 1.66 ppm and 2.5 ppm, respectively.

Phytoplankton

A 81.0-percent decrease in productivity of natural phytoplankton communities occurred when they were exposed for 4 hours to a concentration of 1.0 ppm of folpet (Butler, 1963).

FORMALIN

Fishes

At concentrations in excess of 25 ppm, formalin was toxic to goldfish (Alabama, 1955). The 25-hour LC_{50} for channel catfish to formalin was 87.0 ppm by volume (Clemens and Sneed, 1959).

The 24-hour LC_{50} for striped bass to formalin was 80 ppm (Wellborn, 1969).

Persistence

Formalin applied to soil persisted for <4 days (Domsch, 1963).

α-FURALDEHYDE

Fishes

The 24-hour LC_{50} for harlequin fish to α-furaldehyde was 31 ppm (Alabaster, 1969).

GLYODIN

Mammals

The LD_{50} for the rat was 3,170 mg/kg to glyodin when the mammal was fed the stated dosage orally (FCH, 1970).

Arthropods

Stultz (1955) reported that glyodin applied to orchards in Nova Scotia at recommended rates did little or no harm to any of the principal parasites present in the habitat. Further support of this finding comes from the investigation by MacPhee and Sanford (1961) in the same region; they found that treatments with glyodin at recommended application rates caused little or no reductions in the numbers of beneficial predaceous and parasitic arthropods.

GRISEOFULVIN

Microorganisms

Brian (1949) reported griseofulvin to be effective in reducing the growth of *Zygomycetes*, Ascomycetes, Basidiomycetes, and Fungi Imperfecti at dosages of about 10 μg/ml, whereas it took about 20 μg/ml of griseofulvin to reduce the growth of the Oomycetes.

HEXACHLOROPHENE

Mammals

The LD_{50} for mammals (species not specified) was about 320 mg/kg to hexachlorophene when the mammals were fed the stated dosage orally (FCH, 1970).

Welch (1964) reported that hexachlorophene acted as an effective repellent for small rodents.

HIPPURIC ACID

Fishes

The exposure of brown trout, bluegill, and goldfish to 5 ppm of hippuric acid, a fungicide protectant, for 24 hours resulted in no mortality in any of the species (Spector, 1955).

HYDROXYMERCURICHLOROPHENOLS

Birds

Meadowlarks would not eat seed treated with hydroxymercurichlorophenol (Neff and Meanley, 1956 in Springer, 1957).

Persistence

Hydroxymercurichlorophenol applied to soil persisted for 29 days (Spanis, Munnecke and Solberg, 1962).

LIME SULFUR

Mammals

Lime sulfur applied as a spray or painted on bark proved to be an effective repellent on plants for various species of rabbits, especially the white-tailed jackrabbit (Garlough, Welch and Spencer, 1942).

Birds

No harmful effects on songbirds and their nestlings were observed in orchards treated with lime sulfur (Kelsall, 1950 in Springer, 1957).

Arthropods

The 48-hour EC_{50} (immobilization value at 60°F) for waterfleas, *Simocephalus serrulatus* and *Daphnia pulex*, to lime sulfur was 11 ppm and 10 ppm, respectively (Sanders and Cope, 1966).

Lime sulfur at normal application rates in orchards in Nova Scotia was found to cause significant mortalities to all beneficial species of predators and parasites which occur commonly and play important roles in pest control in orchards (MacPhee and Sanford, 1954). Bartlett (1963) reported similar findings in California. He stated that lime sulfur applied in orchards at a rate of 5.0 gal/100 gal of water was found to be relatively toxic to beneficial parasitic wasps and slightly toxic to beneficial predaceous coccinellids.

MALACHITE GREEN

Mammals

The LD_{50} for the rabbit was 75 mg/kg to malachite green when the mammal was fed the stated dosage orally (PCOC, 1966).

Birds

Dambach and Leedy (1948) reported that malachite green exhibited some promise as a repellent to pheasants.

Fishes

The 24-hour LC_{50} for harlequin fish to malachite green oxalate was 0.46 ppm (Alabaster, 1969).

MALEAMIC ACID

Fishes

The exposure of brown trout, bluegill, and goldfish to 5 ppm of maleamic acid for 24 hours resulted in no mortality in any of the species (Spector, 1955).

MERCURY AND MERCURY COMPOUNDS

Mammals

The LD_{50} for the cat was 100 mg/kg to ethyl mercury p-toluene sulfonanilide (Ceresan M) when the mammals were fed the stated dosage orally (FCH, 1970).

Methoxyethyl mercury is degraded in the bodies of animals to inorganic mercury. Phenyl mercury breaks down in a manner similar to methoxyethyl mercury and is degraded in the bodies of animals to inorganic mercury (Berlin et al., 1969).

Borg et al. (1969) stated that grazing mammals in Sweden, such as roe deer, reindeer, and hares, had negligible mercury (primarily methyl mercury) residues in their bodies; however, mercury levels were relatively high in mammalian predators such as foxes, martens, and polecats. Mercury poisonings were reported frequently for the predators.

Birds

The LD$_{50}$ for young mallards was >2,262 mg/kg; for pheasants, 360 mg/kg; for young coturnix, 668 mg/kg; for pigeons (*Columba livia*), 755 mg/kg; and for prairie chickens, 360 mg/kg to Ceresan M when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970). The LC$_{50}$ for mallards was 30 to 60 ppm; for pheasants, 140 to 160 ppm; and for coturnix, 90 to 110 ppm of Ceresan M in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days, except for the mallards, which were fed for 8 days on clean feed after dosage (Heath et al., 1970).

The LD$_{50}$ for young mallards was >2,000 mg/kg; for young pheasants, 1,190 mg/kg; for young bobwhite quail, 1,060 mg/kg; and for young fulvous tree ducks, 1,080 mg/kg to a formulation containing methylmercury 2,3-dihydroxypropyl mercaptide and methylmercury acetate (Ceresan L) when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Investigations conducted by Nestler and Coburn (1948 in Springer, 1957) demonstrated that grain treated with ethyl mercury phosphate poisoned bobwhite quail in 13 to 20 days. The quail were also found to prefer untreated grain to treated grain.

Leedy and Cole (1950) reported that several mercury fungicides used for seed treatment were quite toxic to pheasants. They quoted the work of Ordal, who listed an LD$_{50}$ of 10 mg/kg for mercury (type not given) fed orally to pheasants. However, grain dressings containing both organomercurials and lindane proved to be non-toxic to both wood pigeons and pheasants in England (Carnaghan and Blaxland, 1957).

Three of 5 pheasants died when fed 10 to 20 grains of corn treated with mercuric phenyl cyanamide (3.6 percent metallic mercury) (Leedy and Cole, 1950).

Meadowlarks ate seeds treated with ethyl mercury phosphate (Neff and Meanley, 1956 in Springer, 1957).

Mercury levels in wood pigeons were determined before 1964 and after (in 1966) when restrictions on the use of mercury for seed dressings were enacted (Wanntorp et al., 1967). Of pigeons shot in 1964, 46.1 percent had residues in their livers exceeding 2 mg/kg, and 20.5 percent had residues exceeding 5 mg/kg. The corresponding levels for birds shot in 1966 were 6.4 percent and 0 percent.

Analyses of the total mercury content in 5 Japanese storks which died at the Obama and Toyooka regions in Japan were found to contain maximum levels in their livers at 61.5 ppm (96.8 ppm in kidney) whereas maximum level in the control little egret liver was 2.1 ppm (Muto and Suzuki, 1967). The direct cause of death of the Japanese storks was not known, but the authors stated that "it was highly possible that they died of chronic poisoning by mercury in diets taken for a long period."

Since the middle 1950's Swedish scientists have documented the widespread mercury (primarily methyl mercury) poisonings in terrestrial animals (Borg et al., 1969). Of 255 seed-eating birds (pheasants, partridges, pigeons, finches, corvine birds) which were found dead and examined, 48 percent had mercury levels above 2 ppm in liver, 30 percent above 5 ppm, 20 percent above 10 ppm, and 13 percent above 20 ppm. Of an equal number of seed-eating birds shot for investigation, the mercury levels were only slightly lower than those found dead. A total of 412 predatory birds (hawks, falcons, buzzards, eagles, owls) were found dead, shot, or trapped for examination. Of these, 62 percent had mercury levels in the liver exceeding 2 ppm, 36 percent exceeding 5 ppm, 19 percent exceeding 10 ppm, and 11 percent exceeding 20 ppm.

When pheasants were fed methyl-mercury-treated wheat (20 ppm) for 9 days, their eggs had reduced hatchability, and residues ranged from 1.3 to 2.0 ppm (Borg et al., 1969).

In the seed-eating birds mercury residues increased significantly in late spring and autumn, indicating a correlation with spring and autumn sowing of treated seed (Borg et al., 1969). These authors also stated that on several occasions game mortality or impaired reproduction could be correlated with mercury poisoning—this was particularly true for the seed-eating birds in dry and/or cold springs.

In the eggs of pheasants and partridges mercury residues averaged about 2.0 ppm in Sweden (Borg et al., 1969).

In laboratory experiments pheasants were fed wheat treated with methyl mercury dicyandiamide about 20 ppm (normal treatment in agriculture) (Borg et al., 1969). The birds died in 29 to 61 days with liver residues ranging from 80 to 180 ppm. Jackdaws fed the same concentration died after 30 to 80 days. Demonstration that the alkyl mercury-treated seed was the main source of animal poisonings came as a result of the discontinuance of mercury seed treatments in 1966.

When chickens were fed methoxy ethyl mercury hydroxide at a dosage of 400 μg per chicken each day, after 7 to 9 days the total mercury in the eggs produced was 0.19 mg/kg (Kiwimäe et al., 1969). Continuing at this same dosage, by days 137 to 139 the level in the eggs had risen to 0.46 mg/kg. Mercury feeding was stopped at this time. Some 39 days later the amount of mercury detected in the eggs was 0.086 mg/kg.

Fishes

The 24-hour LD_{50} for channel catfish to Ceresan M was 1.8 ppm (Clemens and Sneed, 1959).

Plants

When apple trees were sprayed with phenyl mercury acetate ($\frac{1}{16}$ pt/100 gal), the mercury moved in plants by translocation (Ross and Stewart, 1962). Both the new foliage and growing fruit contained mercury. None of the mercury in the soil was taken up through the roots of the apple tree.

Mercury was reported by Lindstrom (1959) to diffuse from the seed coat treated with methyl mercuric hydroxide to the fruit inside. The diffusion was greatly influenced by moisture content of the seed, as indicated by a 300-fold increase in diffusion when the moisture content of the wheat seed was raised from 12 to 18 percent.

Microorganisms

Microorganisms were found to have the capacity to convert inorganic mercury into methyl mercury at a rapid rate (Jensen and Jernelöv, 1969). The methyl mercury was readily taken up by fish. This process of methylation probably explains the uptake and distribution of mercury in the biological system in lakes.

Biological Concentration

Samples of 20 pike caught in a Swedish lake (control) contained 195 to 300 ppb, whereas 20 pike caught in a lake below a pulp mill contained 5,800 to 11,500 ppb of mercury (Hasselrot, 1968).

In fish mercury compounds were taken up directly from the water and from their food (Hannerz, 1968). The rate of concentration was rapid, while the elimination rate was slow, leading to high accumulations in fish (table 54).

TABLE 54. The estimated concentration factors with methoxyethyl mercury, methyl mercury, and mercuric chloride in pike in freshwater (Hannerz, 1968).

Pike Organ	Methoxyethyl Mercury	Methyl Mercury	Mercuric Chloride
Liver	3,000	7,000	1,100
Kidneys	2,000	6,500	1,500
Gills	3,100	8,500	2,700
Muscles	50	800	100

Chinook salmon (2 years old) were fed fingerlings contaminated with 3 ppm of mercury and were found to accumulate mercury in their livers to 30.5 ppm (0.31 ppm, control) and kidneys, 17.5 ppm (1.10 ppm, control) (Rucker and Amend, 1969).

Analysis of mercury content in pike muscle suggested that the biological concentration factor from water to pike is of the order of 3,000 or more (Johnels et al., 1967). A direct relationship between age of pike and mercury content was evident.

No direct association was recorded between the phenylmercuric acetate and methylmercuric hydroxide concentration in animals in the water and the trophic level of the animal in the food chain (table 55).

TABLE 55. The concentration factor of 2 forms of mercury in different organisms and sediment in ponds (Hannerz, 1968).

Organism	Phenylmercuric acetate (20 days, total)	Methylmercuric hydroxide (20 days, total)
	Range	Range
Vegetation	0–4,300	4–5,000
Aquatic worms	2,030	450–1,780
Snails	1,260–1,800	3,400–3,370
Insects	900–12,700	4,100–8,470
Crustaceans	4,370	
Sediment	8,800	8,100

Persistence

A large portion of the organic mercury (ethyl mercury acetate or phenyl mercury acetate) applied to the soil was found to be in the organomercury form after a period of 30 to 60 days. Increasing moisture in the soil caused a decrease in the amount of escaping organic mercury vapor (Kimura and Miller, 1964).

METHIONINE

Fishes

The exposure of brown trout, bluegill, and goldfish to 5 ppm of methionine (medicinal) for 24 hours resulted in no mortality in any of the species (Spector, 1956).

METIRAM

Fishes

The 24-hour LC_{50} for rainbow trout to metiram was 22 ppm (Alabaster, 1969).

Arthropods

Ulrich (1968) reported that metiram remaining on a surface after treatment with a concentration of 1,000 ppm in water killed at least 88 percent of adult female *Trichogramma* with a 10-hour exposure.

MYSTOX

Fishes

The 24-hour LC_{50} for rainbow trout to mystox LSL/L, LSL/P, LSE/L, and LSE/P was 330, 60, 68, and 47 ppm, respectively (Alabaster, 1969).

NABAM

Mammals

The LD_{50} for the rat was 395 mg/kg (FCH, 1970) and for the domestic goat, >500 mg/kg (Tucker and Crabtree, 1970) to nabam when the mammals were given the stated dosages orally in a capsule.

Birds

The LD_{50} for young mallards was >2,560 mg/kg; for young pheasants, 707 mg/kg; for young coturnix, 2,120 mg/kg; and for pigeons (*Columba livia*), >2,000 mg/kg to nabam when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970). The LC_{50} for mallards was >5,000 ppm; for pheasants, >5,000 ppm; and for coturnix, >5,000 ppm of nabam in diets of 2-week-old birds when fed treated feed for 5 days followed by clean feed for 3 days (Heath et al., 1970a).

Fishes

The 24-hour LC_{50} for channel catfish to nabam was 21.1 ppm (Clemens and Sneed, 1959).

Mollusca

Nabam at concentrations from 0.5 ppm to 10 ppm prevented clam eggs from developing (Davis, 1961).

Arthropods

Stein (1961) reported that nabam at recommended dosages (1 to 10 lb/A in Thomson, 1967) caused significant (about 50-percent) reductions in parasitization of pest insect eggs by *Trichogramma*, a parasitic wasp.

Adult female *Trichogramma* exposed to nabam at a dosage of 1,000 ppm as a residue were killed (at least 80 percent) within 4 hours (Ulrich, 1968).

Microorganisms

From nabam a volatile toxicant, carbonyl sulfide, was produced, and this material was found to be lethal to soil fungi (Moje, Munnecke and Richardson, 1964).

Persistence

Nabam applied at 100 ppm to soil persisted for >20 days (Domsch, 1958).

NALCO

Fishes

The 24-hour LC_{50} for harlequin fish to nalco 240, 201, and 243 was 9 ppm, 0.8 ppm, and 0.33 ppm, respectively (Alabaster, 1969).

NAPHTHALENSULFONIC ACID

Fishes

The exposure of brown trout, bluegill, and goldfish to 5 ppm of naphthalensulfonic acid (protectant material) for 24 hours resulted in no mortality in any of the species (Spector, 1955).

ORTHOZID

Arthropods

Stein (1961) reported that orthozid caused significant reductions (about 80 percent) in parasitization of pest insect eggs by *Trichogramma*.

OXYTETRACYCLINE

Fishes

The 24-hour LC_{50} for striped bass to oxytetracycline was >250 ppm (Wellborn, 1969).

PARA-DICHLOROBENZENE

Mammals

The LD_{50} for rats was 1,000 to 4,000 mg/kg to para-dichlorobenzene when the mammals were fed the stated dosages orally (PCH, 1970).

Fishes

The 48-hour LC_{50} for rainbow trout exposed to para-dichlorobenzene was 880 ppb (FWPCA, 1968).

PCNB

Mammals

The LD_{50} for the rat was >12,000 mg/kg to PCNB when the mammal was fed the stated dosages orally (PCH, 1970).

Microorganisms

Caseley and Broadbent (1968) reported that there was little or no influence on soil nitrification with PCNB with dosages ranging from 10 to 1,000 ppm.

Persistence

After 10 months 80 percent of 5 mg of PCNB applied to 50 g of soil was lost (Caseley, 1968). Increasing the moisture level of the soil significantly increased the rate of PCNB loss from soil.

PHENETIDINE

Fishes

The exposure of brown trout, bluegill, and goldfish to 5 ppm of phenetidine (protectant material) for 24 hours resulted in no mortality in any of the species (Spector, 1955).

PHENOXYTOL

Fishes

The 24-hour LC_{50} for harlequin fish to phenoxytol was 160 ppm (Alabaster, 1969).

PMA

Mammals

The LD_{50} for the rat was 100 mg/kg to PMA when the mammal was fed the stated dosage orally (FCH, 1970).

Fishes

The 24-hour LC_{50} for rainbow trout to PMA was 0.036 ppm (Alabaster, 1969).

POTASSIUM PERMANGANATE

Fishes

The 96-hour LC_{50} for striped bass to potassium permanganate was 2.5 ppm (Wellborn, 1969).

PRB

Arthropods

PRB at a concentration of 0.15 percent was reported by Van de Vrie (1962) to cause 80-percent mortalities in 7 days to predatory mites (*Typhlodromus tiliae* and *T. tiliarum*).

PROPINEB

Mammals

The LD_{50} for the rat was 8,500 mg/kg to propineb when the mammal was fed the stated dosage orally (FCH, 1970).

Arthropods

Commercial fungicide formulations of propineb at 0.15 percent proved harmless to a common wasp parasite, *Aphytis holoxanthus* (Rosen, 1967).

QUININE

Fishes

The 96-hour LC_{50} for channel catfish to quinine sulfate was 42.0 ppm (Clemens and Sneed, 1959).

Amphibians

The lethal dose of quinine to frogs by subcutaneous injection was 200 to 400 mg/kg (Spector, 1955).

SAFROLE

Fishes

The exposure of brown trout, bluegill, and goldfish to 5 ppm of safrole (medicinal) for 96 hours resulted in no mortality for the bluegill and goldfish, but 100-percent mortality for the brown trout (Dittmar, 1950).

SALICYLIC ACID

Amphibians

The median lethal doses of salicylic acid to frogs by subcutaneous injection was 500 to 900 mg/kg (Spector, 1955).

SMDC

Mammals

The LD_{50} for the male rat was 820 mg/kg to SMDC when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LC_{50} for pheasants was >5,000 ppm of SMDC in diets of 2-week-old birds when fed treated feed for 5 days followed by untreated feed for 3 days (Heath et al., 1970).

Fishes

The 24-hour LC_{50} for harlequin fish to SMDC was 0.19 ppm (Alabaster, 1969).

Annelids

No earthworms of either *Lumbricus* or *Helodrilus* survived in pots containing soil treated with 50, 100, and 400 gal/A of SMDC (DeVries, 1962).

Persistence

SMDC applied to soil persisted for 1 hour (Gray, 1962).

SODIUM NITRITE

Fishes

The 24-hour LC_{50} for harlequin fish to sodium nitrite was 880 ppm (Alabaster, 1969).

SULFUR

Mammals

Hayne (1949) reported that wettable sulfur at 10 tablespoons in 1 qt of water applied as a heavy coating to beans and cabbages repelled cottontail rabbits.

Arthropods

Wettable sulfur applied to orchards in Nova Scotia at normal application rates was found to cause significant reduction (about 75 percent) in the numbers of beneficial parasitic and predaceous species (MacPhee and Sanford, 1954).

Sulfur at normal application rates in orchards in Nova Scotia were reported by Stultz (1955) to reduce seriously the numbers of predators (*Haplothrips faurei*, *Leptothrips mali*, *Anystis agilis*, and *Anthocoris musculus*) attacking the bud moth. Wasp parasites *Meteorus trachynotus* and *Ascogaster quadridentata* were only rarely seen after several years' use of flotation sulfur at normal application rates (Stultz, 1955); however, the parasite *Agathis laticinctus* functioned effectively in the treated orchards.

Wettable sulfur was found to cause only slight mortality to the adults of the parasite *Aphelinus mali* (Schneider, 1958).

Further support of the above came from the investigation of MacPhee and Sanford (1961), who reported that the use of wettable sulfur at recommended dosages in orchards in Nova Scotia reduced the numbers of beneficial parasites and predators of some of the serious apple pests. Bartlett (1963) also reported that sulfur applied to orchards in California at a rate of 5.0 lb/100 gal of water was highly toxic to some beneficial wasps, but less toxic to beneficial predaceous coccinellid beetles.

Wettable sulfur at a concentration of 0.25 percent in 7 days killed 90 percent of the exposed predatory mite populations (*Typhlodromus tiliae* and *T. tiliarum*) (Van de Vrie, 1962). A similar finding (high mortalities of predaceous mites) was reported by Besemer (1964) when wettable sulfur was applied at recommended dosages to orchards.

In later investigations by Van de Vrie (1967), wettable sulfur applied to orchards at a concentration of 0.25 percent caused little or no harm to the predatory bug *Anthocoris nemorum*, but was toxic to the parasite *Aphelinus mali* and highly toxic to the predatory bug *Orius* sp.

Plants

The effect of sulfur added to the soil annually at 1,250 lb/A for 1949 and 1950 was measured by growing various crops plants in the contaminated soil during 1954 and 1955 (MacPhee, Chisholm and MacEachern, 1960). With high residues in the soil of sulfur at the time of growth, yields of the crop plants were as follows: beans, reduced by 55 percent; turnips, little effect; carrots, reduced by 100 percent; tomatoes, little effect; and peas, little effect.

TETRACYCLINE HYDROCHLORIDE

Fishes

The 96-hour LC_{50} for striped bass to tetracycline hydrochloride was >1,018 ppm (Wellborn, 1969).

THIOUREA

Amphibians

The median lethal dosage of thiourea to frogs by subcutaneous injection was 10,000 mg/kg (Spector, 1955).

Persistence

Thiourea applied at 200 ppm to soil persisted for 10 to 20 weeks (Jensen and Bendixen, 1958).

THIRAM

Mammals

The LD_{50} for the rat was 780 mg/kg to thiram when the mammal was fed the stated dosage orally (FCH, 1970).

Thiram applied at a rate of 2½ oz per bushel of seed corn reduced the acceptance by white-footed mice of the treated corn by 40 percent, compared with untreated corn (Welch and Graham, 1950). Thiram also has been reported to repel field and harvest mice and rabbits (Welch, 1954; Mann, Derr and Meanley, 1956 in Springer, 1957). A 7.27-percent thiram concentration was reported by Hildreth and Brown (1955) to repel rabbits.

Birds

The LD_{50} for young mallards was >2,800 mg/kg and for young pheasants, 673 mg/kg to thiram when the birds were given the stated dosage orally in a capsule (Tucker and Crabtree, 1970).

Thiram has been found to repel birds (Welch and Graham, 1950; Welch, 1954; Mann, Derr and Meanley, 1956 in Springer, 1957), but treated seed corn had little or no effect when fed to pheasants (Leedy and Cole, 1950).

When thiram was included at 10 to 200 ppm in the diets of chickens, it caused the chickens to produce soft-shelled and abnormally shaped eggs (Waibel, Pomeroy and Johnson, 1955). The majority of the eggs were soft-shelled when the chickens received feed with 100 ppm of thiram. These findings were confirmed by Antoine (1966), who also found that thiram caused chickens to produce abnormal eggs. In addition, he reported that chicks fed 50 ppm of thiram in their diet had a noticeable weakness in their legs.

Fishes

The 72-hour LC_{50} for channel catfish to thiram was 0.79 ppm (Clemens and Sneed, 1959).

Arthropods

Thiram applied to orchards at recommended rates was reported by Schneider (1958) to have no effect on the parasite *Aphelinus mali*. At a concentration of 0.15 percent, thiram was reported to kill 70 percent of a predatory mite population (*Typhlodromus tiliae* and *T. tiliarum*) after 7 days of exposure in the laboratory (Van de Vrie, 1962). Besemer (1964) working in the field, however, reported that normal application rates of thiram did not harm predatory mites in orchards.

Thiram at 1.6 percent concentration (normal spray concentration is 0.16 percent) resulted in a 5-percent mortality to the parasite *Mormoniella* (Ankersmit et al., 1962). There was no significant difference between the control population and treated wasp populations.

Van de Vrie (1967) reported that thiram applied to apple orchards at a 0.15-percent concentration caused little or no mortality to two predatory bugs (*Anthocoris nemorum* and *Orius* sp.), but did cause some mortality to the parasite *Aphelinus mali*.

Thiram remaining on a surface after treatment with a concentration of 1,000 ppm in water had little or no effect upon *Trichogramma* female adults exposed for 10 hours (Ulrich, 1968).

Microorganisms

Thiram soil treatment at 50 ppm changed the microbiological balance in soil; the number of bacteria increased, while the number of fungi declined (Richardson, 1954). Thiram was selective in its action against fungi, with *Penicillium* and *Trichoderma* being resistant and increasing in number with time.

Persistence

Thiram applied at a rate of 50 ppm in sandy soil was found to persist for over 2 months (Richardson, 1954).

When thiram was well distributed in soil it showed extremely low persistence (Griffith and Matthews, 1969). Under these conditions the fungicide had a half-life of 1 to 2 days. However, when this material was applied in heavy concentrations on the surface of beads (simulating seeds), thiram persisted extremely well; there was little change in concentration even after 21 days.

THYMOL

Amphibians

The lethal dose of thymol to frogs by subcutaneous injection was 150 mg/kg (Spector, 1955).

TRIAMIPHOS

Mammals

The LD_{50} for the male rat was 20 mg/kg triamiphos when the mammal was fed the stated dosage orally (FCH, 1970).

Arthropods

Van de Vrie (1962) reported that triamiphos at a concentration of 0.05 and 0.10 percent caused little or no mortality to predatory mite populations (*Typhlodromus tiliae* and *T. tiliarum*). Remmer (1964) also reported that triamiphos as a single application in orchards caused no mortality to *T. tiliae* and *T. tiliarum* populations.

When apple orchards were treated with triamiphos at a concentration of 0.10 percent, little or no mortality was recorded in the predatory bug *Anthocoris nemorum* population, but high mortalities occurred in the predatory bug *Orius* sp. and parasite *Aphelinus mali* populations (Van de Vrie, 1967).

About 50 percent of a population of adult female *Trichogramma* were killed within 10 hours after being exposed to triamiphos remaining on a surface after treatment at a concentration of 250 ppm in water (Ulrich, 1968).

TRIBUTYL TIN OXIDE

Fishes

The 24-hour LC_{50} for rainbow trout to tributyl tin oxide was 0.027 ppm (Alabaster, 1969).

ZINC HYDROXYQUINONE

Fishes

The 24-hour LC_{50} for harlequin fish to zinc hydroxyquinone was 0.17 ppm (Alabaster, 1969).

ZINEB

Mammals

The LD_{50} for the rat was 5,200 mg/kg to zineb when the mammal was fed the stated dosage orally (FCH, 1970).

Birds

The LD_{50} for mallards was >2,000 mg/kg and for young pheasants, >2,000 mg/kg to zineb when the birds were given the stated dosages orally in a capsule (Tucker and Crabtree, 1970).

Arthropods

The use of zineb at recommended dosages in orchards in Nova Scotia caused little or no reduction in the numbers of beneficial predatory and parasitic arthropods (MacPhee and Sanford, 1961). Also, zineb and dithiocarbamate mixtures with maneb at recommended dosages in orchards were generally harmless to an egg parasite (*Trichogramma* sp.) under field conditions (Besemer, 1964). Zineb was reported to have no effect on the parasite *Aphelinus mali* at normal rates of application in orchards (Schneider, 1958).

Mites appear to be sensitive to zineb (Van de Vrie, 1962); he reported that a 0.10-percent concentration would kill about 70 percent of predatory mite populations (*Typhlodromus tiliae* and *T. tiliarum*) after 7 days of exposure. In field applications of zineb at a concentration of 0.20 percent, little or no mortality was observed in the predatory bug populations (*Anthocoris nemorum* and *Orius* sp.), but some mortality was recorded in the parasite *Aphelinus mali* population (Van de Vrie, 1967).

Persistence

Zineb applied to soil persisted for ≥75 days (Domsch, 1958).

ZIRAM

Mammals

The LD_{50} for the rat was 1,400 mg/kg to ziram when the mammal was fed the stated dosage orally (FCH, 1970).

Ziram was found to repel rabbits and woodchucks, but caused no mortality to these and other animals when used at recommended dosages (Welch, 1951 in Springer, 1957). Ziram also was reported as being effective in repelling small rodents (Welch, 1954).

Birds

Ziram was reported to be harmless to birds when used at recommended dosages (Welch, 1951 in Springer, 1957). Ziram also has been reported not to deter blackbirds from eating rice (Neff and Meanley, 1955 in Springer, 1957).

Fishes

The 96-hour LC_{50} for channel catfish to ziram was 1.0 ppm (Clemens and Sneed, 1959).

Persistence

Ziram applied to soil persisted for ≥55 days (Domsch, 1958).

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PART V

Pesticide Residues in the Environment

Literature on pesticide residues and their movement in the ecosystem has been carefully selected to provide a general view of the environmental problem.

Mammals

During 1961 Durham et al. examined beaver, caribou, moose, polar bear, seal, walrus, and whale for DDT and DDE, but found no trace in these animals in Alaska.

Holden and Marsden (1967) on the Atlantic coast in Canada found harp seals to contain dieldrin at 0.07 ppm, DDE at 3.5 ppm, TDE at 0.78 ppm, and DDT at 3.5 ppm. Also, off the coast of Scotland the grey seal, common seal, and porpoise contained significant levels of both DDT and dieldrin. The mean residues recorded in one group of 18 seals were: dieldrin at 0.79 ppm, DDE at 8.8 ppm, TDE at 1.8 ppm, and DDT at 7.8 ppm. The mean for 3 porpoises was dieldrin at 9.9 ppm, DDE at 12.8 ppm, TDE at 8.9 ppm, and DDT at 91.0 ppm.

In the Antarctic Sladen, Menzie and Reichel (1966) reported traces of DDT in the crab-eater seal. Fat from Weddell seals, also collected in the Antarctic, contained residues of DDT and DDE ranging from 0.005 to 0.105 ppm in the seals (Brewerton, 1969). During the 3-year period of sampling there was no indication of an increase in residue concentrations.

Harp seal milk from a seal in the Gulf of St. Lawrence was analysed as having the following residues: DDE, 0.47 ppm; DDT, 0.58 ppm; and TDE, 0.11 ppm (Cook and Baker, 1969).

Fur seals were collected on Pribilof Islands, Alaska, in 1968 and off the Washington coast in 1967. Of the 30 seals examined, all contained DDE; 21, TDE; 24, DDT; and 5, dieldrin (Anas and Wilson, 1970). Also, in the livers of sick and dying immature California sea lions, DDE residues were found at concentrations of 4.0 and 89.0 ppm (Haderlie, 1970).

Adipose tissue sampled from 350 big game animals in 1962 revealed residues of DDT, DDE, and TDE (table 66).

Pronghorn antelope were examined for aldrin, dieldrin, endrin, lindane, heptachlor, and DDT, TDE, plus DDE combined (Moore, Greichus and Hugghins, 1968). The 45 animals examined contained the following residues: lindane, 0.04 to 0.08 ppm; heptachlor epoxide, 0.04 to 0.12 ppm; and combined DDT, TDE, plus DDE, 0.06 to 0.17 ppm.

Residues from 0.5 to 2.8 ppm of DDT and its metabolites were found to persist in mink up to 9 years after 2 single applications of DDT at 1 lb/A to Maine forests (Sherburne and Dimond, 1969). Wild hares were shown to have low residue levels (0.02 ppm) for up to 10 years after spraying. In the control or untreated plot hares had average residue levels of 0.01 ppm of DDT.

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Caribou fat averaged 0.1 ppm of organochlorine compound, whereas polar bear fat averaged about 25 times higher (Keith, 1969).

TABLE 66. Average residues in adipose tissue of big game animals (Walker, George and Maitlen, 1965)

Animal	Samples (Number)	Average Residues (ppm) TDE	DDE	DDT (isomers)[1]
		State of Idaho		
Antelope	4	≤0.01	≤0.01	0.098
Bear	5	≤0.01	≤0.01	0.032
Deer	67	≤0.01	0.01	0.109
Elk	44	≤0.01	0.03	0.071
Goat	1	≤0.01	≤0.01	0.050
Moose	3	≤0.01	0.01	0.087
		State of Washington		
Bear	13	≤0.01	≤0.01	0.045
Deer	109	≤0.01	0.01	0.122
Elk	82	≤0.01	0.04	0.058
Goat	9	≤0.01	≤0.01	0.023

[1] Combined ortho, para' and para, para' DDT.

Birds

In Alaska during 1961 Durham et al. found no trace of either DDT or DDE in the eider duck, but found 1.1 ppm of DDE in 2 white owls.

Moore and Walker (1964) in England reported that the birds having the highest concentration of organochlorine insecticide residues were fish-feeding birds, followed in order by raptorial, omnivorous, and herbivorous terrestrial birds (figure 1). Further support of these findings comes from Ratcliffe's (1965) studies, in which he reported that raptor eggs contained 5.3 ppm of organochlorine residues, whereas corvids contained only 0.9 ppm (table 67).

Moore and Tatton (1965) detected residues of DDE and dieldrin in the eggs of 17 species of sea birds in England. The total residues were of approximately the same order and ranged from 0.4 to 5.5 ppm, but there was some indication that the birds feeding on larger fish had higher residues.

Osprey eggs taken from the Connecticut River habitat contained 6.5 ppm of DDT, and 7 species of fish analyzed from this river contained DDT in amounts ranging from 0.8 to 8.1 ppm (wet weight basis) (USDI, 1965).

In Ireland several species of birds and birds' eggs were found to contain residues of mercury and chlorinated insecticides (table 68).

TABLE 67. The organochlorine residues (ppm) in eggs of raptor and corvid birds. Figures in parentheses are the number of nests from which eggs were analyzed (Ratcliffe, 1965).

Raptors		Corvids	
Peregrine falcon (13)	13.8	Raven (8)	2.1
Merlin (3)	6.2	Carrion crow (14)	0.8
Golden eagle (7)	2.9	Hooded crow (1)	0.8
Buzzard (4)	2.5	Rook (10)	0.4
Kestrel (4)	1.0	Magpie (3)	0.4

TABLE 68. Range of organic mercury and chlorinated residues in birds and bird eggs (Eades, 1966).

(All results expressed in ppm on a wet weight basis)

Species	Number of Specimens Analyzed: Total	Number With Mercury	Number With Insecticide	Mercury	Lindane	Aldrin	Dieldrin	pp'DDT
Adults:								
Pheasant	11	3	0	2.33-5.15	0.025-0.58	0.01-0.20	0.03-12.80	0.03-0.68
Kestrel	1	0	0	[2] ND	ND	ND	ND	ND
Thrush	2	0	0	ND	ND	ND	ND	ND
Pigeon	3	3	3	0.015-5.33	0.025-0.07	0.02-0.04	0.005-0.10	0.08-0.19
Bullfinch	1	0	1	ND	0.22-0.28	0.18-0.20	0.70-1.80	0.36-0.56
Eggs:								
Mallard	[1] 6	0	5	ND	0.01-0.015	0.002-0.01	0.06-0.135	0.01-0.04
Guillemot	[1] 5	1	5	0.57	0.003-0.04	0.003-0.06	0.25-0.44	0.07-0.46
Fulmar	[1] 4	0	3	ND	0.03-0.04	0.01	0.33-0.44	0.41-0.56
Razorbill	[1] 6	0	5	ND	0.01-0.045	0.01-0.09	0.30-0.43	0.46-0.72
Kittiwake	[1] 5	1	4	0.61	0.02-0.04	0.01-0.02	0.29-0.46	0.32-0.55

[1] [illegible] analyzed only for organic mercury residues.
[2] ND, none detected.

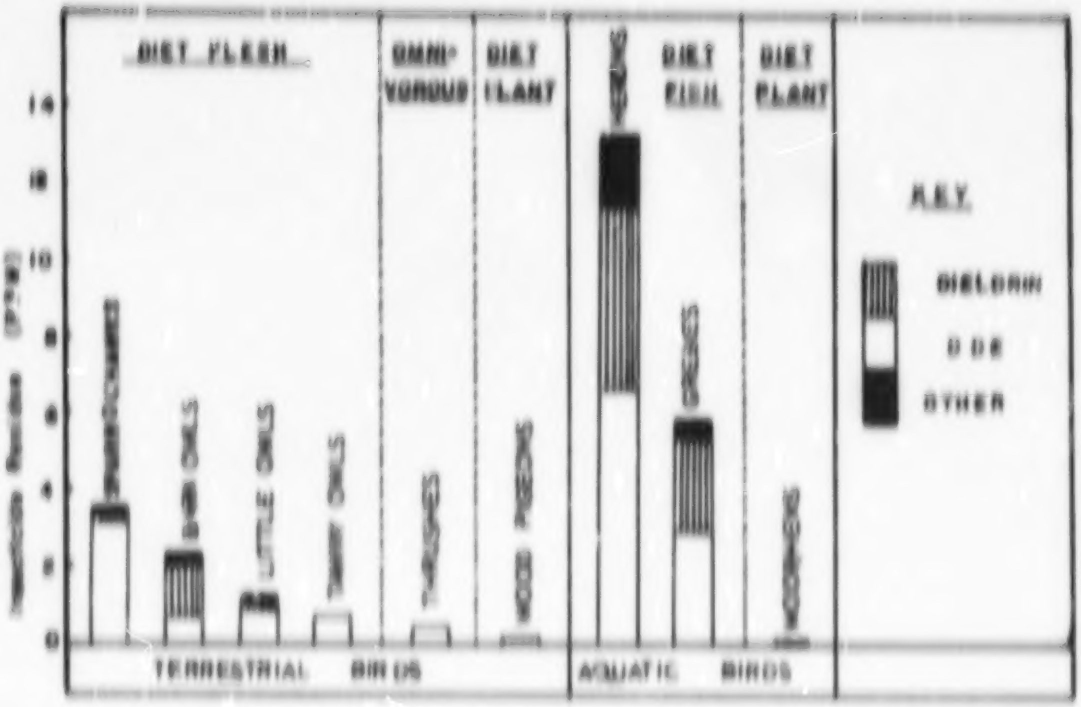

FIGURE 1. Average concentration of organochlorine insecticide residue in the breast muscle of different types of birds. The following were investigated (number of specimens analyzed given in brackets): sparrow hawk (8), barn owl (9), little owl (7), tawny owl (5), thrush (4), wood pigeon (8), heron (7), great crested grebe (4), and moorhen (6) (Moore and Walker, 1964).

From 1880 to 1940 the mercury levels in Sweden in the white-tailed eagle were significantly higher (about 6,000 ng/g in feathers) than in the peregrine falcon (about 2,000 ng/g in feathers) (Berg et al., 1966). This difference was due to differences in food habits. After 1940 mercury levels in birds increased 10 to 20 times or more because of increased use of mercury in Sweden. Analysis of "normal" game and "fallen" game for mercury in Norway showed residues below 1 mg/kg for 73 percent of "normal" and 71 percent of "fallen" game (Holt, 1969). Predaceous birds had higher levels of mercury than did the prey. Of the wood pigeons, about 78 percent had a mercury content below 1 mg/kg, and only 7 percent above 2 mg/kg. The corresponding percentages for birds of prey were 55 and 28.

Residues of DDT were found in the Antarctic in 4 of 16 Adelie penguins and 12 of 16 skuas (*Stercorarius*) (George and Frear, 1966). The one emperor penguin did not contain DDT. The maximum insecticide residues (wet weight) were 0.18 ppm of DDT in Adelie penguins and 2.8 ppm in skuas.

Traces of DDT, BHC, heptachlor, and dieldrin were found in chinstrap penguins in Antarctica (Tatton and Ruzicka, 1967). Samples of fat collected from 6 Adelie penguins and analyzed for DDT and its metabolites contained 24 to 152 ppb of DDT. The control consisted of a flipper from an emperor penguin that had been frozen for 52 years in Antarctica (previous to the use of DDT) (Sladen, Menzie and Reichel, 1966).

White pelicans which had died in widely scattered locations in western United States were analyzed for insecticide contamination (USDI, 1965). Composite residue samples were as follows: DDT, 194 ppm; toxaphene, 82 ppm; and dieldrin, 10 ppm.

DDT residues in herring gulls, old-squaw ducks, and ring-billed ducks in the same habitat of Lake Michigan varied greatly and were 99 ppm, 28 ppm, and 6 ppm, respectively (Hickey, Keith and Coon, 1966). Food habits were proposed as the cause of these differences (Dustman and Stickel, 1969).

In northwestern Lake Michigan a sample of 9 apparently alive herring gull eggs contained averages of 202±34 ppm (wet weight) of DDE. The 10 dead eggs sampled had higher concentrations of 219±117 ppm of DDE. Of 115 nests examined in this study, an exceptionally high number of the eggs, 30 to 35 percent, were dead (Keith, 1966). In another study herring gull eggs on Lake Michigan were found to contain 227 ppm of DDT and its metabolites, and adult birds contained 99 ppm in breast muscle and 2,441 ppm in fat (Keith, 1966, and Hickey, Keith and Coon, 1966). Four hen pheasants from an intense agricultural area in California were found to have the following residues in their fat: DDT, 1,236 to 2,930 ppm; DDE, 309 to 717 ppm; and dieldrin, 0.1 to 28 ppm (Hunt and Keith, 1963). Egg yolks from this area contained 106 to 1,080 ppm DDT, 23 to 161 ppm DDE, and 0 to 1.3 ppm dieldrin.

In a small sample of only 4 bald eagle eggs collected in Maine, New Jersey, and Florida from 1964 to 1965, the eggs contained 6 to 16 ppm of DDT and its metabolites, and 0.5 to 1.0 ppm of dieldrin (Stickel et al., 1966). These amounts were similar to those found in British peregrine falcon eggs which averaged 12.4 ppm of DDE, 0.8 ppm of dieldrin, and 1.4 ppm of other chlorinated insecticides (Moore and Walker, 1964). The eggs from Scottish golden eagles had 0.5 to 7.0 ppm of dieldrin (Ratcliffe, 1964).

Double-crested cormorant eggs from habitats associated with the lakes of Wisconsin, Minnesota, North Dakota, Manitoba, and Saskatchewan were found to contain 11 ppm of chlorinated insecticide residues, primarily DDT and its metabolites (USDI, 1966). White pelican eggs from the habitat contained only 2.4 ppm residues.

The average levels of insecticide residues in pheasants in California were higher than those in any other species of wildlife: DDT, 57.82 (0.00 to 3,768) ppm; DDE, 68.20 (0.15 to 2,680) ppm; TDE, 0.01 ppm; and dieldrin, 0.84 ppm (Keith and Hunt, 1966). Birds of prey contained up to about 60 ppm of DDT and its metabolites plus low levels of dieldrin, endrin, heptachlor epoxide, and toxaphene. Song birds, such as mountain chickadees, from a forested area which had received DDT had levels of DDT as high as 21 ppm. Keith and Hunt reported that shore birds had relatively high (10 to 70 ppm) levels of DDT. Fish-eating birds like the white pelican contained high levels of DDE (39 ppm in fat) plus lower levels of other insecticides.

DDT (DDT, DDE, and TDE) residues in sea birds resident in California ranged from 0.7 ppm in the brain of Cassin's auklet to 911 ppm in the fat of the Western gull (Risebrough et al., 1967). Non-resident sea birds were found to contain about the same average amount of DDT and its metabolites.

The overall mean residues of chlorinated insecticides found from 1963 to 1966 in peregrine falcon eggs in Scotland were as follows: DDE, 15.7 ppm; DDT, 0.1 ppm; TDE, 0.1 ppm; dieldrin, 0.7 ppm; heptachlor epoxide, 0.9 ppm; and BHC, 0.3 ppm (Ratcliffe, 1967).

In the Peace, Slave, and MacKenzie Rivers in Canada the fat of 9 adult female peregrine falcons had average DDT, DDE, TDE, dieldrin, and heptachlor epoxide residues of 87.5, 984, 59.5, 8.8, and 4.4 ppm (wet weight), respectively (Enderson and Berger, 1968). Immature peregrine falcons, however, caught in Wisconsin have averaged only 0.9, 14.0, 0.8, 0.2, and 0.0 ppm (wet weight) of the same materials. Residues of chlorinated insecticides (DDT, DDE, TDE, dieldrin, and heptachlor epoxide) in peregrine falcon prey averaged about 1.0 ppm (wet weight whole body).

In adult peregrine falcon fat Aroclor (PCB), dieldrin, and DDT plus its metabolites were found to be 1,080 ppm, 50 ppm, and 2,600 ppm, respectively (Risebrough et al., 1968b).

Peregrine falcons were found to have residues of DDT and its metabolites plus dieldrin, 617.0 ppm in fat to 5.30 ppm in brain (wet weight), higher than those of the small birds, 0.20 ppm to 2.05 ppm in whole body (wet weight), on which the falcons fed (Cade, White and Haugh, 1968).

Wurster and Wingate (1968) reported DDT residues averaging 6.44 ppm in eggs and chicks of the carnivorous Bermuda petrel.

Stickel and Stickel (1969) reported that residues of DDT were lower in the bodies of dead birds than in survivors that had received the same dosage of DDT for the same period of time. DDT in dead males was found to average 74 ppm, whereas in the survivors DDT content averaged 171 ppm (significant, $P < 0.05$). DDT in dead females averaged 55 ppm and in surviving females averaged 128 ppm (too few birds for significance).

Chlorinated insecticide residues in starlings were examined at 128 sites throughout the United States (Martin, 1969). DDT and its metabolites were found in all samples taken, and the residues ranged from <0.1 to 0.8 ppm. Other insecticide residues in order of level of contamination were heptachlor epoxide, lindane, and BHC. Highest residue levels were found in the following regions: Southeastern U.S., southern New Mexico, Arizona and California, eastern Utah, and the Willamette River drainage in Oregon.

A total of 45 bald and 21 golden eagles found sick or dead in 18 states and Canada during 1964 and 1965 were analyzed for pesticide residues (Reichel et al., 1969). The median residues in the bald eagle for 1964 and 1965, respectively, were as follows: *p,p'*-DDE, 7.90 ppm and 8.00 ppm; *p,p'*-TDE, 1.60 ppm and 0.44 ppm; *p,p'*-DDT, 0.49 ppm and 0.20 ppm; dieldrin, 0.65 ppm and 0.84 ppm; heptachlor epoxide, 0.09 ppm. The golden eagles, however, had only a trace of these chemicals, except for DDE (0.49 ppm).

In the nationwide program to monitor pesticides in the wings of 24,000 mallard and black ducks during 1965 and 1966, DDE was shown to be the predominant residue, followed in order by DDT, TDE, dieldrin, and heptachlor epoxide. Residues were highest in the Atlantic and Pacific Flyways and lowest in the Central Flyway. DDE was the highest in the ducks from New Jersey, Massachusetts, Connecticut, Rhode Island, New York, Pennsylvania, Alabama, California, and Utah. Dieldrin residues were also prevalent in wings from Arkansas, Texas, Utah, California, and several states along the Atlantic Flyway (Heath, 1969).

In a survey of eggs from aquatic prairie habitats, waterfowl eggs averaged about 2 ppm of organochlorine residues, while eggs from gulls and other fish-eating birds ranged between 2 and 20 ppm (Keith, 1969). Atlantic gannets feeding on mackerel and herring in Canadian populations also were contaminated with organochlorine insecticides, and their eggs contained between 8 and 100 ppm.

Western grebes collected on the Tule Lake National Wildlife Refuge had DDT residues ranging from 0.07 to 995 ppm (fat) in one part of the marsh and 58 to 1,293 ppm (fat) in another part of the marsh (Keith, 1969). The investigator suggested that these differences reflected variation in exposures and ability to retain these residues.

White pelican eggs and double-crested cormorant eggs collected in central North America during summer of 1965 averaged 1.7 ppm and 10.4 ppm (wet weight) of DDE (Anderson et al., 1969). Arochlor averaged 0.6 ppm and 8 ppm in the 2 bird species, respectively. Significant correlations were found between shell thickness (and weight) and both DDE and Arochlor in cormorant eggs.

Average residues of dieldrin in dead birds collected in Britain during 1964 were as follows: sparrow hawk, 1.69 ppm; kestrel, 0.56 ppm; barn owl, 0.60 ppm; tawny owl, 0.11 ppm; insectivorous bird species, 0.5 ppm; woodland bird species, 0.1 ppm; heron (*Ardea cinera*), 6.7 ppm; and freshwater non-fish-eating bird species, 1.0 ppm (Robinson, 1969). Note the high residues in aquatic birds and especially in the fish-eating heron species.

Fat from Adelie penguins collected in the Antarctic contained residues of DDT and DDE ranging from 0.045 to 0.77 ppm (Brewerton, 1969). During the 2-year sampling there was no indication of a buildup of residues.

Lockie, Ratcliffe and Balharry (1969) reported that the proportion of golden eagle eyries in west Scotland which successfully reared young increased from 31 percent in the period 1963-65 to 69 percent in the period 1966-68. At the same time the level of dieldrin in eagles' eggs fell from 0.86 ppm (1963-65) to 0.34 ppm (1966-68). During these periods the number of sheep carcasses per 10-mile transect remained the same; however, there was a decrease in dieldrin used in sheep dips, and this resulted in a decrease in dieldrin residues in mutton fat on which the eagles fed. During 1964 the mean residue in mutton fat was 0.8 ppm (0.0 to 12.4 ppm); during 1965 the mean residue was 1.1 ppm (0.0 to 8.2 ppm); and during 1966 the mean residue was 0.4 ppm (0.0 to 5.8 ppm).

Aldrin, dieldrin, DDT, DDE, TDE, heptachlor epoxide, lindane, and endrin residues were found in pheasant adults, juveniles, and eggs collected in South Dakota (Linder and Dahlgren, 1970). Only 2 adults were found with more than 1 ppm of any one insecticide. All the juveniles contained residues of at least one insecticide. Dieldrin and heptachlor epoxide occurred more often than the other insecticides in the eggs. Only one egg was found with 1.58 ppm dieldrin.

Along a stretch of beach on Monterey Bay 440 birds were found dead (Haderlie, 1970). Of these, 37 percent had died of oiling, 14 percent had been shot, and 49 percent had died of unknown causes. The latter group was found to contain in their livers the following DDE residues: Brandt's cor-

cormorants, 107 to 135 ppm; Western grebes, 192 to 289 ppm; fork-tailed petrel, 378 ppm; Ashey petrel, 375 ppm; and ring-billed gull, 805 ppm.

Arochlor residues were found in the fat of brown pelicans collected on Anacapa Island (California) ranging from 77 to 380 ppm (Keith, Woods and Hunt, 1970).

Mercury concentrations in one great blue heron collected at Lake St. Clair were 175 ppm in liver and 23 ppm in carcass (Dustman, Stickel and Elder, 1970). Maximum levels in a common tern were 33 ppm in liver and 7.5 ppm in carcass. Mercury levels in fish removed from the stomachs of 3 great blue herons contained 3.5, 1.5, and 3.0 ppm, and a fish from the common tern contained 3.3 ppm. Mercury levels in the breast muscle of waterfowl exceeded 0.5 ppm in 4 of 8 mallards, in one of 4 blue-winged teal, and in all 4 lesser scaup.

Fishes

A total of 16 species of fish collected from New York waters contained from 0.2 to 7.0 ppm of DDT (wet weight) (Mack et al., 1964). Some tissues, such as visceral fat, gills, eggs, and reproductive organs, had residues as high as 40 ppm. No residues were found of aldrin, dieldrin, lindane, chlordane, heptachlor, or endrin.

Fifteen months after a farm pond was treated with 0.02 ppm of DDT the residues in trout were essentially the same as immediately after treatment; also, residues in bullheads did not decline significantly during the same period (Bridges, Kallman and Andrews, 1963).

In the eggs of chinook salmon off the shore of California residues of DDT and its metabolites were found up to 666 ppb (Modin, 1969). DDT and metabolites were found in halibut up to 591 ppb.

Residues of DDT averaging 0.44 ppm were found in the Antarctic in 8 samples of 3 fish species (George and Frear, 1966).

Most species of fish sampled in California by Keith and Hunt (1967) contained residues of DDT, DDE, TDE, dieldrin, endrin, heptachlor epoxide, aldrin, and toxaphene. A few species had quite high levels; for example, the fat of the white catfish contained 145.80 ppm of DDT, 275.22 ppm of DDE, 106.57 ppm of TDE and 8.08 ppm of dieldrin.

DDT or its metabolites were detected in 100 percent of all fish taken in Wisconsin or the boundary waters (Kleinert, Degurse and Wirth, 1968). The concentration of DDT and its analogs of TDE and DDE averaged 27.15 ppm and ranged from 0.520 to 134.6 ppm in fat. Nearly 70 percent of fish collected had dieldrin residues. These residues averaged 0.15 ppm and ranged from 0.026 to 870.2 ppm in fat.

Of the 590 composite whole fish samples collected in the Great Lakes, 584 contained DDT and other metabolites, with levels ranging up to 45 ppm (mg/kg wet weight, whole fish) (Henderson, Johnson and Inglis, 1969). Dieldrin was found in 75 percent of the samples, with levels ranging up to nearly 2 ppm. Other organochlorine insecticide residues were found in a few of the samples; some of these had high residue levels.

The insecticide levels in edible fish of the Pacific Northwest monitored by Stout (1968) were as follows: anchovy, DDE at about 74 ppb, and TDE at about 55 ppb; English sole, DDT at about 15 ppb, DDE at about 15 ppb, and TDE at about 18 ppb; hake, DDT at about 100 ppb, DDE at about 60 ppb, and TDE at about 60 ppb; ocean perch, DDT at 15 ppb, DDE at 10 ppb, and a trace of TDE; starry flounder, DDT at 15 ppb, DDE at 18 ppb, and TDE at 26 ppb; true cod, DDT at 4 ppb, DDE at 5.5 ppb, and TDE at 5.5 ppb; and yellowtail rockfish, DDT at about 50 ppb, DDE at about 100 ppb and TDE at about 50 ppb.

Fish from Lake Michigan were found to contain residues of DDT and dieldrin 2 to 7 times those in fish from the other Great Lakes (Reinert, 1970). Average concentrations of DDT and dieldrin in only a few of the fish species sampled are shown in table 68. Reinert also showed a high correlation between percentage fat and size fish, and these 2 characters were in turn highly correlated with DDT content in the fish.

Fish species collected from British waters contained the following quantities of arsenic (As_2O_3): whiting, 0.01 ppm; plaice, 0.08 ppm; sole, 0.07 ppm; hake, 0.02 ppm; herring, 0.01 ppm; cod, 0.01 ppm; haddock, 0.01 ppm; brill, 0.01 ppm; mackerel, 0.02 ppm; halibut, 0.03 ppm; and turbot, 0.04 ppm (Cox, 1925).

Largemouth bass collected in the Rock River in Iowa contained arsenic (As_2O_3) which ranged from 0.100 ppm to 1.80 ppm (Wiebe, Gross and Slaughter, 1931). These levels were considerably lower than those reported by the Swedish Arsenic Commission, which recorded the content of cod to range from 0.5 ppm to 4.1 ppm (average 1.8 ppm).

TABLE 29. Average concentrations of DDT (DDT, DDE, and TDE) and dieldrin in a few selected fish species from the Great Lakes (Reinert, 1970)

Lake	Fish Species	DDT ppm	Dieldrin ppm
Michigan	Lake trout	6.96	0.20
"	Yellow perch	0.23	0.06
Ontario	American smelt	1.06	0.10
"	Yellow perch	0.10	0.008
Huron	American smelt	0.76	0.04
"	Yellow perch	1.09	0.03
Erie	Walleye	1.12	0.14
"	Yellow perch	0.87	0.05
Superior	Coho salmon	1.00	0.01
"	Lake trout	7.44	0.05

Amphibians

DDT residue persistence was investigated in Maine forests which had been treated with 1 lb/A for spruce budworm control (Dimond et al., 1968). Residues (0.1 to 0.8 ppm) were found to persist in the red-backed salamander population for 6 to 8 years after the DDT treatment.

Molluscs, Arthropods, and Annelids

Analyses of pesticides in oysters, mussels, and clams in California estuaries indicated DDT, TDE, DDE, dieldrin, and endrin in concentrations ranging from 10 to 5,000 ppb (Modin, 1969).

DDT residues in the flesh of shellfish off the coast of California were generally low (Keith and Hunt, 1966). The common Washington clam, common little neck clam, and Pacific oyster contained 0.05 ppm, 0.28 ppm, and 0.17 ppm of DDT, respectively.

Trichoptera and Plecoptera collected in a stream in southern Sweden below a paper mill were found to contain maximum levels of mercury of 17,000 ng/g and 6,600 ng/g, respectively (Johnels et al., 1967). Above the mill Trichoptera and Plecoptera contained maximum mercury levels of 74 ng/g and 70 ng/g, respectively.

In offshore samples DDT, TDE, and DDE were found in king crab up to 6,700 ppb (Modin, 1969).

The insecticide levels in edible Dungeness crab of the Pacific Northwest were DDT, trace to 0.013 ppm; DDE, 0.027 to 0.040 ppm; and TDE, 0.017 to 0.031 ppm (Stout, 1968).

In a Missouri refuge earthworms contained from 0.29 to 1.101 ppm of dieldrin, even though the fields had not been treated since 1963 (USDI, 1966). In a private cotton field in Alabama the earthworms contained "dangerously high levels of endrin, 3.40 and 3.60 ppm."

Worms, slugs, and snails were collected for residues from heavily treated cotton and corn fields on National Wildlife refuges in Mississippi and Missouri (USDI, 1966). From the Mississippi refuge the sample contained 40 ppm of DDT + TDE, 1.14 ppm of endrin, and 0.45 ppm of dieldrin in slugs. These levels would be toxic to several animal species if eaten as the prime source of food. Earthworms in this region appeared to pick up little dieldrin and endrin, but contained as much as 90 ppm of DDT + TDE.

From 67 fields of 16 crop types in 8 states, samples of soils and earthworms were obtained and analyzed for residue (dry weight) (USDI, 1966). On an average there was about 9 times as much organochlorine residue load in the earthworms as in soil. About 17 percent of the fields had earthworms with residues of 20 ppm or greater. The highest levels were from earthworms containing total organochlorine residues of 159, 110, 116, and 109 ppm. In the sample DDT + TDE were recorded as 155, 90, 33, and 90 ppm; dieldrin ranged from 0.08 to 82.5 ppm; and endrin, a maximum of 2.7 ppm. Based on previous investigations, these levels were reported hazardous to birdlife.

Davis and Harrison (1966) examined soil invertebrates in arable soil habitats and apple orchards for chlorinated insecticides. They reported that samples of beetles contained both dieldrin and p,p' DDE in amounts up to 0.[illegible] and 2.5 ppm, respectively. There was no obvious difference between beetle samples from the 2 sites. Dieldrin ranged from 2 to 10 times higher in earthworms from the orchard site than in those from some of the arable soil sites and ran generally higher in the earthworms than in the beetle samples. Earthworms from the orchard site contained 11.7 to 30.4 ppm DDT, and slugs contained from 5.5 to 28.8 ppm.

Boykins (1966) on the Michigan State campus collected soil and earthworms from an American elm habitat treated with DDT (elms sprayed with a 12 percent DDT solution) and found the following residues in soil and earthworms: in spring 1963 the soil had 30.1 ppm and earthworms had 67.7 ppm; and in spring 1964 the soil had 31.0 ppm, *Lumbricus terrestris* had 62.9 ppm, and *Helodrilus caliginosus* had 64.8 ppm.

Risebrough et al. (1967) analyzed several species of marine invertebrates for DDT (DDT, DDE, and TDE) and found that they contained from 6 ppb in the purple urchin to 165 ppb in short-spined purple snail.

Whitstable oysters collected off the coast of Sweden were found to contain as much as 3.7 ppm of arsenic (As_2O_3) (Cox, 1925).

Several species of earthworms were analyzed for chlorinated insecticides in an English soil (table 90). Residues of the insecticides were consistently higher in the smaller, more shallow-living species, *Allolobophora caliginosa*, *A. chlorotica*, and *A. rosea*, than the deep-living species, *Lumbricus terrestris*, *A. longa*, and *Octolasion cyaneum*.

Residues of chlorinated insecticides in beetles and earthworms from English soil on which had been grown winter wheat and peas are shown in table 91. In most cases both the beetles and earthworms had higher concentrations of the insecticides in their bodies than did the soil.

Soil invertebrates from 67 agricultural fields in 8 different states were examined for chlorinated insecticides (DDE, TDE, DDT, aldrin, dieldrin, endrin, heptachlor epoxide, and gamma chlordane) (Gish, 1970). Overall insecticide residues averaged 1.5 ppm (dry weight) in soil and 13.8 ppm (dry weight) in earthworms; in general, residue levels in earthworms averaged 9 times those in soil. The residues in soils ranged from a trace to 19.1 ppm and in earthworms, from a trace to 159.4 ppm. The author also reported that residues in beetle larvae from 2 fields averaged 0.6 ppm, snails from 2 fields averaged 3.5 ppm, and slugs from 2 fields averaged 33.0 ppm.

Plants

In Alaska Durham et al. (1961) examined cranberries, salmonberries, and wild rhubarb for DDT and DDE, but found no trace of insecticide in these Eskimo foods.

Kale plants in pots were placed on plots which had been treated with either aldrin or dieldrin at 9.5 ppm each in soil and were found to contain either aldrin (0.18 ppm in leaf) or dieldrin (0.04 ppm in leaf) (Walker, 1966). The author concluded that the evidence strongly documented the aerial transfer of aldrin and dieldrin to kale leaves.

Plants from 3 wildlife habitat types were examined for DDT and DDE residues (Keith and

TABLE 90. Concentrations (ppm) of chlorinated insecticide residues in 6 species of earthworms in relation to the residue content of the soil (Wheatley and Hardman, 1968).

	Aldrin	Dieldrin	p,p'-DDT	o,p'-DDT	p,p'-DDE	Lindane
Soil	0.73	0.64	0.83	0.13	0.17	0.004
Earthworm species:						
Lumbricus terrestris	0.053	1.6	0.54	0.080	0.53	0.0084
Allolobophora longa	0.25	2.2	0.77	0.10	0.38	0.0085
Allolobophora caliginosa	0.54	3.6	1.5	0.23	0.65	0.011
Allolobophora chlorotica	0.68	4.8	2.3	0.72	1.0	0.013
Allolobophora rosea	0.64	3.9	1.6	0.25	0.72	0.017
Octolasion cyaneum	0.44	2.4	0.67	0.10	0.38	0.0078

TABLE 91. Residues (ppm) at one site in soil, beetles, and earthworms (Davis, 1968).

	Lindane	Aldrin	Dieldrin	p,p'-DDT	o,p'-DDT	p,p'-TDE	p,p'-DDE
Soil		0.03	0.1	0.3	0.04	0.03	0.1
Beetles:							
Harpalus sp.	0.1	NS	0.05	NS	NS	NS	2.2
Agonum sp.	NS	NS	0.15	NS	NS	NS	0.06
Earthworms	0.05	0.02	0.5	2.1	0.2	0.2	0.9

NS = not sampled.

Hunt, 1966). In untreated marshes the pondweed contained 0.78 ppm of DDT and 0.33 of DDE. The upland habitat which had received 1 lb/A of DDT annually had in the grasses 28.8 ppm of DDT and 0.14 ppm of DDE and in the shrubs 80.0 ppm of DDT and 0.15 ppm of DDE. The forest habitat which had received one treatment with ½ lb/A of DDT had residues of 7.10 ppm in grasses, 2.46 ppm in forbs, 4.13 ppm in sagebrush, and 2.06 ppm in fir foliage.

The leaves of field-grown corn were reported to have higher levels (0.70 to 0.98 ppm) of dieldrin than greenhouse-grown corn (0.04 to 0.15 ppm) (Barrows et al., 1969). The large increase in amount of dieldrin found in the field-grown corn was attributed to aerial contamination of the foliage.

Phytoplankton collected in Monterey Bay, California, from 1955 to 1969 contained *p,p'*-DDT, *p,p'*-TDE, and *p,p'*-DDE in increasing concentrations (Cox, 1970). The level of DDT and its metabolites in 1955 was about 0.20 ppm and increased yearly to about 0.55 ppm in 1969.

Soil

Of each half-pound of DDT released by plane, about 0.2 pound of DDT reaches the forest soil (Woodwell, 1961). He predicted that the persistence of the residues in the forest soil at the low application rate is fewer than 10 years. Soils from DDT-sprayed forests revealed an increase in residues between 1958 and 1961, although no further treatments were made. The suggestion is that the DDT residues present in the tree canopies were slowly carried into the soil (Woodwell and Martin, 1964).

Approximately 40 percent of the DDT applied to the test orchards since 1948 was still present in the soil, mostly in the top 12 inches (Terriere et al., 1966).

In a preliminary study of the residues present in soil in the Mississippi River Delta area, low levels of DDT, endrin, calcium arsenate, aldrin, BHC-lindane, strobane-toxaphene, and heptachlor were detected. Interestingly enough, most of these chlorinated insecticides were also found in well water, although residue levels were lower than that of the soil (USDA, 1966).

The application of aldrin and heptachlor in 5 yearly dosages of 5 lb/A resulted in 1.7 to 2 times higher soil insecticide residues than those soils receiving one 25 lb/A treatment (Lichtenstein, 1966). The 5-application treatment resulted in residue levels of 4.6 lb/A (18 percent of total applied dosage) at the end of the 5-year period. The one massive dose application resulted in residue levels of only 10 percent of the applied dosage 5 years later.

Repeated applications (4 to 5 applications) resulted in residue levels which were about 20 percent of the total applied dosages (1, 2, or 4 lb/A). Of the crops grown, beet, radish, cucumber, lettuce, turnip, celery, carrot, potato, parsnip, broccoli, and in some cases cabbage did not absorb measurable amounts of the insecticides from the soil (Lichtenstein and Schulz, 1965).

From 2 terrestrial wildlife habitats the soil in the upland habitat which had been treated annually with 1 lb/A of DDT contained 2.03 ppm of DDT and 0.10 ppm of DDE, and soil in the forest habitat which had been treated once with ½ lb/A of DDT contained 0.40 ppm of DDT and 0.18 ppm of DDE (Keith and Hunt, 1966). The litter in the forest habitat had 7.00 ppm of DDT and 0.28 ppm of DDE.

Water

Toxaphene recovered from water in a drainage basin in Alabama from the summer of 1959 through the fall of 1960 had concentrations ranging from 0.029 to 0.14 ppb. In the same drainage basin mean seasonal recoveries of BHC ranged from 0.022 to 0.18 ppb (Grzenda, Lauer and Nicholson, 1964).

Insecticide contamination was measured in a stream draining from a 400-square-mile watershed area in northern Alabama, where about 15,000 acres of cotton are grown annually (Nicholson et al., 1964). Toxaphene, BHC, and DDT, comprising over 90 percent of the insecticides used, were estimated at 58,000 pounds in 1960 and 130,000 pounds in 1962. Tests in the stream water showed that toxaphene ranged from 0.007 to 0.41 ppb, and BHC ranged from 0.007 to 1 ppb; DDT was never detected.

During 1961 and 1962 water in the Columbia Basin Project in southeastern Washington was sampled for pesticides (Hindin, May and Dunstan, 1964). Aldrin (0.04 to 2.0 ng/l), DDT (0.02 to 18 ng/l), TDE (0.4 ng/l), endrin (0.4 to 57 ng/l), and 2,4-D (isooctyl ester and butyl ester, trace to 28 ng/l) were found in the sampled water.

Low levels of insecticides were detected in 80 percent of 82 water samples from marshes, irrigation canals, streams, rivers, and lakes in California (Keith and Hunt, 1966). The insecticide and residue levels measured were as follows: DDT and its metabolites, 0.02 ppm; BHC, 0.01 ppm; toxaphene, 0.02 ppm; dieldrin, trace; methoxychlor, 0.09; and heptachlor epoxide, trace.

Parathion-treated duck ponds, one receiving 1.0 and the other 0.1 pound per acre, contained 0.46 to 0.51 ppm of parathion in the water immediately after treatment. The level decreased to 0.01 ppm 8 days after treatment and to 0.003 ppm 14 days after. Parathion residues never exceeded 0.08 ppm in the mud, measured 4 hours to 32 days after the treatment (Mulla, Keith and Gunther, 1966).

Carbaryl, applied at the rate of 25 ml per liter, persisted for 17 days in seawater at 20°C; Karinen et al. (1967) proposed that in mud carbaryl was likely to persist for 2 to 6 weeks.

In a 7-year illustrative summary of the occurrence of dieldrin, endrin, and the DDT group in major river basins in the United States, dieldrin dominated the pesticide picture during the period from 1958 to 1964 (Breidenbach et al., 1967).

From Cypress Creek, Tennessee, sediment samples revealed extremely high residues of the following insecticides: isodrin, 12,000 ppm; aldrin, 3,000 ppm; endrin, 10,000 ppm; dieldrin, 9,000 ppm; and chlordane, 30,000 ppm (Barthel et al., 1970).

Typical estuaries with positive analysis residue levels were in the range of 10 to 200 $\mu g/kg$ for DDT, DDE, or TDE (Butler, 1969). Dieldrin and endrin residues were also common in a few estuaries. Because some residues were in the range of 10 to 20 $\mu g/g$ in fish and oysters, Butler undertook experiments to determine effects on fish and crustacean of a DDT-contaminated diet. Dietary levels of 2.5 $\mu g/g$ of p,p' DDT were found to cause 35 to 100 percent mortality within 2 to 10 weeks in laboratory populations of shrimp, crabs, and fish.

In an estuary flowing through a truck-farming agricultural area, there were 2 distinct peaks in the level of DDT found in the estuary, one about April and the other in December. These 2 were correlated with the 2 harvest times that took place annually in the region (Butler, 1969).

The residues of pesticides in estuaries isolated from agricultural areas seldom were above 100 ppb, whereas in agricultural regions residues were found as high as 11,000 ppb in shell fish (Modin, 1969).

Levels of pesticides and/or residues were measured from 1965 to 1968 in selected western streams (Manigold and Schulze, 1969). Aldrin, TDE, DDE, DDT, dieldrin, endrin, heptachlor, heptachlor epoxide, and lindane were detected at one time or another. DDT was the most common insecticide, with a maximum concentration of 0.12 $\mu g/l$.

Seba and Corcoran (1969) reported concentration factors in sea slicks of chlorinated insecticides up to 100,000-fold above the level in the sea water.

Residues of TDE were followed downstream of the St. Lawrence River when it was treated during 1966 and 1967 with a total of 36,831 lb of technical TDE for control of nuisance insects (Fredeen and Duffy, 1970). Residues detected in water (up to 0.0130 ppm) ranged from 1 to 17 percent of the amount applied to the river 10 miles upstream. TDE residues in snails (*Campeloma* sp.) and bivalves (*Pisidium* sp.) 17 miles upstream from the application point and 10 and 45 miles downstream averaged 0.002, 0.101, and 0.0, respectively. Five species of fish at the same location had TDE residues of 0.150 ppm (17 miles upstream) and 0.360 ppm (combined downstream).

Air

Concentrations of DDT associated with suspended particulate matter in Pittsburgh air during 1964 ranged from 0.80 to 1.99 $\mu g/1000\ mm^3$ (Antommaria, Corn and De Maio, 1965).

Air over both rural and urban communities in the United States was shown to contain pesticides (Tabor, 1965 in Cohen and Pinkerton, 1966). Concentrations of DDT were found to range from below detectable levels to 23 ng/m^3 for rural air samples and from below detectable levels to as much as 8,000 ng/m^3 for urban communities which had pest control programs. The author pointed out that these concentrations were minimal because only an unknown portion of the particulate matter and none of the pesticide in vapor form was captured by the techniques employed.

The amount of pesticide released into the atmosphere above the crop, but which does not reach the treated crop, is well illustrated with aircraft spraying. For example, in treating corn by aircraft Hindin, May and Dunstan (1966) reported that only about 26 percent of the DDT sprayed from aircraft during 2 seasons reached the corn when measured at tassel height.

Weibel et al. (1966) reported that of 80 rainwater samples analyzed, none was free of organochlorine, which ranged from 0.02 to 1.18 ppb. The insecticides detected and the town in Ohio where the rainwater samples were collected are as follows: DDT at Ripley (0.15 ppb), Coshocton (0.07 ppb), and Cincinnati (0.34 ppb); DDE at Ripley (0.03 ppb), Coshocton (0.005 ppb), and Cincinnati (0.02 ppb); and BHC at Ripley (0.05 ppb), Coshocton (0.006 ppb), and Cincinnati (0.02 ppb).

The data of Wheatley and Hardman (1965) from Central England showed rainwater collected during November to February contained p,p'-DDT 0.003 ppb, dieldrin 0.020 ppb, and lindane 0.100 ppb, whereas samples collected during January to March contained 0.003, 0.008, and 0.020 ppb, respectively. Also, analyses of the atmosphere in Britain revealed that BHC, dieldrin, DDE, TDE, and DDT were all present in the atmosphere. The levels ranged from 0.010 ppb for BHC to 0.400 ppb for DDT. These materials occurred as vapor or by occlusion on dust particles and were "scrubbed-out" by rain and snow from the atmosphere (Abbott et al., 1965).

Abbott et al. (1966) also reported finding via silicone gas-liquid chromatography the following pesticides in the air of London: lindane, 0.005 ppb; dieldrin, 0.018 ppb; p,p'-DDT, 0.003 ppb; p,p'-DDE, 0.004 ppb; and p,p'-TDE, 0.003 ppb.

Dust collected in the atmosphere over Cincinnati was found to contain the following concentrations of pesticides: DDT, 0.6 ppm; chlordane, 0.5 ppm; DDE, 0.2 ppm; ronnel, 0.2 ppm; heptachlor epoxide, 0.004 ppm; 2,4,5-T, 0.04 ppm; and dieldrin, 0.003 ppm (Cohen and Pinkerton, 1966).

Residues of pesticides were detected in substantial quantities in the air over various communities in southeastern United States (table 92).

In a later investigation by Tarrant and Tatton (1968) at 7 collecting stations on the British Isles, the mean concentrations of organochlorine pesticides for the year at all stations were: lindane at 0.065 ppb; dieldrin, 0.008 ppb; p,p'-DDE, 0.002 ppb; p,p'-TDE, 0.014 ppb; and p,p'-DDT, 0.51 ppb.

Chlorinated hydrocarbons in the air were found moving on dust particles with the Trade Winds from the European-African land areas to the Barbados range (Risebrough et al., 1968a). The total concentration of DDT, DDE, TDE, and dieldrin ranged from 1 to 164 ppb.

TABLE 92. Quantities of pesticides in the air of various communities (Tabor, 1966)

Insecticide	Type Community	Residues ng/m³	
		Minimum	Maximum
DDT	Urban	(1)	430
Malathion	Urban	(1)	140
DDT	Suburban	0.5	8,500
Chlordane	Suburban	1	31
DDT	Agricultural	0.4	22
Chlordane	Agricultural	0.3	2.2
Toxaphene	Agricultural	1.2	7.5
Thiophosphates	Agricultural	(1)	(1)

[1] Trace.

In an investigation of the pesticides in rainfall conducted by the Geological Survey (1969) following concentrations were recorded in the Florida Everglades region: TDE, 0.00 to 20.7 ppb; DDE, 0.00 to 21.5 ppb; DDT, 0.00 to 28.5 ppb; dieldrin, 0.00 to 0.01 ppb; lindane, 0.00 to 0.07 ppb; and aldrin, endrin, and heptachlor below detectable levels.

Ambient air was sampled in 1967 at 9 locations throughout the United States (Barney, 1970). 2,4-D was detected at Salt Lake City, Utah, at 4.0 ng/m³; 2,4,5-T was also detected in 1970 in air samples (Yobs, 1970).

Barney (1970) sampled ambient air in 1967 for pesticides at Baltimore, Md.; Buffalo, N.Y.; Dothan, Ga.; Fresno, Calif.; Iowa City, Iowa; Orlando, Fla.; Riverside, Calif.; Salt Lake City, Utah; and Stoneville (no state mentioned) and found DDT (4.8 to 2,080 ng/m³), DDE (0 to 141 ng/m³), BHC (0 to 22 ng/m³), lindane (0 to 7 ng/m³), heptachlor (0 to 19 ng/m³), aldrin (0 to 8 ng/m³), toxaphene (0 to 2,520 ng/m³), dieldrin (0 to 30 ng/m³), endrin (0 to 58 ng/m³), parathion (0 to 465 ng/m³), methyl parathion (0 to 129 ng/m³), and malathion (0 to 2 ng/m³). The "highest levels were found in cotton soybeans agri-area of the Mississippi Delta." The study has been continuing and Barney reports that generally the levels detected in 1970 have been higher than those in the previous study. In addition to those detected earlier, diazinon and endosulfan were also found (Yobs, 1970).

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PART VI

An Evaluation and Summary

Of the nearly 1 billion pounds of pesticides applied in the United States during 1970, about 51 percent was for farm use, and the remaining 49 percent for public and governmental use. This amounts to about 5 lbs of pesticide applied per person for pest control. The bulk of the pesticides was aimed at about 2,000 pest species; these species make up only about 1 percent of the total 200,000 species of plants and animals in the United States. As expected, many of the non-target species were directly or indirectly affected by the pesticides used.

In the encyclopedic review of the ecological effects of pesticides on non-target species, there is wide variation in the amount of information available concerning the effects of a particular pesticide. DDT, for example, when compared with other pesticides, has been well investigated, as have some others in the chlorinated insecticide group. Even so, the available data on the impact of these pesticides involve fewer than 1,000 species of the estimated total of 200,000 species. The abundance or scarcity of information on a particular pesticide should not be interpreted as an indication of either a hazard or the absence of one. In general, a quick scan of the data reveals that the greatest amount of information is available on insecticides and the least amount on fungicides and their effects on non-target species. Information concerning effects of herbicides is intermediate, yet nearly as much herbicide material is applied as insecticides.

Modes of Action of Pesticides

Little is known concerning the mode of action of most pesticides for either pest or non-target species. The available evidence documents the fact that the mode of action of each pesticide varies significantly with individual species. For example, DDE (a metabolite of DDT) is practically nontoxic to insects, but predaceous bird species like the American sparrow hawk are highly sensitive to it. This chemical affects the predaceous birds' reproductive physiology and causes the birds to produce eggs with eggshells from 10 to 30 percent thinner than normal. Interestingly enough, seed-eating birds like quail and pheasants are relatively resistant to the effects of DDE.

Reducing Species Numbers

The direct application of pesticides to crop lands, forests, and other habitats may reduce and sometimes temporarily exterminate not only the pest, but also non-target species in the treated area. This, of course, is not surprising because a pesticide is an active poison applied specifically to destroy animals and plants designated as pests.

While the direct effects of pesticides are relatively easily observed, the indirect effects are far more complicated to detect. For example, it is difficult to discern whether the numbers of a species are declining and if they are, whether the decline is because of a pesticide or because of the numerous other environmental factors which impinge upon natural populations. In investigating the indirect effects of pesticides it may be difficult to determine how the pesticide was transported in the environment, how the non-target species were exposed, and what dosage of pesticide they received.

An example of the problems involved in determining the impact of pesticides on non-target species was the investigation of why some predaceous bird species declined in habitats where chlorinated insecticide residues were abundant. Some wildlife biologists suspected DDT residues were having an adverse effect, but the influence of urbanization was recognized as an additional factor contributing to bird mortality. When studying natural populations, it is nearly impossible to single out each factor and gauge just how it contributes to the total mortality.

Proof that DDT was responsible for the observed decline of some predaceous birds required the exposure of some of these birds to known amounts of pesticides under controlled conditions. To do this the investigators first had to rear birds of prey (in this case the American sparrow hawk) in the laboratory and then feed them measured amounts of DDT and dieldrin at dosages similar to those occurring naturally. Feeding a combination of DDT and dieldrin in the diet of the sparrow hawk caused the birds to produce eggs with significantly thinner eggshells, and the loss of eggs was significantly increased above untreated controls.

Another example of measuring the impact of a pesticide indirectly on a non-target species involved the decline of lake trout in Lake George and other nearby lakes. For several years previous to the observed decline in the lake trout population, about 10,000 pounds of DDT had been applied yearly for pest control in the watershed surrounding Lake George. Some DDT found its way into the lake, but the amount was believed to be small. Although DDT residues were found in both adult lake trout (8 to 838 ppm of DDT in fat) and their eggs (3 to 355 ppm of DDT), the mature lake trout appeared unaffected, and their eggs hatched normally. The reason for the decline remained a mystery until it was discovered that the young fry were highly sensitive to certain levels of the DDT in the eggs. Thus fry were killed at the time of final absorption of the yolk and just when the young were ready to feed. With 3 ppm of DDT in the eggs a few fry survived, but at 5 ppm or higher mortality was 100 percent. The reason the lake trout population was declining in the lakes was then obvious.

Predaceous and parasitic insect populations have been reduced and even eliminated in some regions after insecticide usage, and this sometimes resulted in outbreaks of particular insect and mite species which had been previously kept under control by these species. For example, when predaceous coccinellid beetle populations were inadvertently eliminated in areas treated with DDT, chlordane, and other chemicals, outbreaks of mites, aphids and scale insects occurred. At times the densities of these plant pests increased 20-fold above their "natural control" level.

Habitat Alteration and Species Reductions

Man using plow and bulldozer has significantly altered many natural habitats and caused significant reductions in some species of plants and animals, but pesticides have been equally effective in altering habitats. Dimethoate applied to a red clover field, for example, reduced the number of insects on which mice were feeding, and this reduced the numbers of mice present. DDT and other insecticides which find their way into streams significantly reduce invertebrate populations. Subsequently, salmon and other fish populations which depend upon these invertebrates may also be reduced.

Herbicide destruction of plants on which animals depend for food may also cause significant reductions in their numbers. For example, 2,4-D applied to a gopher habitat reduced the forbs by 83 percent, and eventually this resulted in an 87-percent reduction in the dependent gopher population.

Changes in vegetation are usually detrimental to dependent species, but the change may also be favorable for other species. For instance, when the tops of mature forest trees were killed with herbicides, the trees sprouted from their bases, thus improving the browse for white-tailed deer.

Behavioral Changes

Pesticides have been found to alter the normal behavior of several animal species. For example, sublethal dosages of dieldrin fed to sheep increased the number of trials required by the animals to relearn a visual discrimination test. Also, sublethal doses of DDT caused trout to lose most of their learned avoidance response.

Salmon, when exposed to sublethal doses of DDT, were found to prefer water of higher temperatures than usual. If this type of exposure occurred in nature, the salmon might place their eggs in regions where their fry could not survive. Mosquito fish exposed to low concentrations of DDT (0.1 to 20 ppb) tended to prefer waters with a higher level of salinity than normal for the species.

The herbicide 2,4-D caused predaceous coccinellid beetles to be sluggish; and this change would alter their predatory activities and effectiveness as a biological control agent.

Growth of Animals and Plants

The biological activity of pesticides suppressed growth in some species and stimulated growth in others. Female white-tailed deer, for example, when fed 5 ppm and 25 ppm of dieldrin daily in their diet for 3 years grew much more slowly than untreated females.

2,4-D was reported to increase the time for growth and development of predaceous coccinellid beetle larvae by nearly 60 percent. This could significantly reduce the effectiveness of these animals in biological control of aphids and other pest insects. On the other hand, 2,4-D stimulated the growth of the rice stemborer pest. Caterpillars of the borer grew 45 percent larger on the treated plants than on the untreated rice plants.

Plant growth may also be affected, as when corn and beans were grown in soil treated with DDT at 10 ppm and 100 ppm. At the end of 4 weeks the corn weighed nearly 40 percent more than corn in the untreated soil. Beans, however, weighed significantly less (30 percent) after 8 weeks when exposed to DDT concentration of 10 ppm than beans grown in untreated soil.

Reproduction

Pesticides caused measurable changes in the reproduction of various non-target animals. White-tailed deer females fed 25 ppm of dieldrin in their food, for example, had lower fawn survival than untreated does.

Pesticides appear to have a deleterious effect on the reproduction of some predaceous birds such as the American sparrow hawk, already mentioned. In some natural habitats the brown pelican has been exposed to DDT and DDE, and it is reported that egg breakage has resulted recently in a complete reproductive failure. Generally, aquatic fish-eating birds have been more severely affected than terrestrial-bird predators because they obtain more DDT, DDE, and other pesticide residues in their food.

Unfortunately, the effects of pesticides on reproduction in birds are more varied than just eggshell thinning. For example, ovulation time in finches reportedly doubled when the birds were fed DDT in their diet, thus increasing the time required for a generation. Also, embryo mortality during egg incubation ranged from 30 to 50 percent when mallard ducks had been fed 40 ppm of DDE in their diet. Total duckling production was reduced by as much as 75 percent when the ducks received this level of DDE.

DDT, DDE, and dieldrin were not the only chemicals to affect reproduction in birds. Both 2,4-D and 2,4,5-T at relatively high dosages depressed reproduction in chickens, as did 2,4-D and silvex in mallard ducks and toxaphene in bobwhite quail and pheasants, and thiram-exposed chickens produced abnormally shaped and soft-shelled eggs.

Female mosquito fish reproduction was affected because they aborted their young after surviving exposure to sublethal dosages of DDT, TDE, methoxychlor, aldrin, endrin, toxaphene, heptachlor, and lindane.

Pesticides may also increase the rate of reproduction. For example, the exposure of bean plants to 2,4-D increased aphid progeny production during a 10-day period from 130 to 764 per aphid mother.

Food Quality Changes

The chemical makeup of plants may be altered by pesticides, and this in turn affects the dependent animals. The changes which do occur appear to be quite specific for both plants and pesticides. Several chlorinated insecticides both increased some elements and decreased others in corn and beans. For example, heptachlor in soil at dosages of 1, 10, and 100 ppm caused significant changes in the macro and micro elements (N, P, K, Ca, Mg, Mn, Fe, Cu, B, Al, Sr, and Zn) measured in the aboveground portions of corn and bean plants. Zinc was significantly higher (89 ppm, dry weight) in bean plants treated with 100 ppm of heptachlor than in untreated controls (55 ppm); however, nitrogen levels were significantly lower (4.99 percent) in the treated plants than in the untreated controls (7.25 percent). Investigators reported an increased protein content in wheat exposed to 2,4-D in contrast with beans grown on 2,4-D-treated soil, which reduced protein content in the beans.

The potassium nitrate content of sugar beet plants exposed to a sublethal dosage of 2,4-D increased from a normal of 0.22 percent to 4.5 percent (dry weight), a nitrate level highly toxic to cattle. 2,4-D and other herbicides have also been reported as increasing the nitrate content of various other plants.

Another change in the chemical content of plants after exposure to pesticides is an increase in sugars. Ragwort, a weed naturally toxic to many animals including cattle, when exposed to sublethal doses of 2,4-D, has a high level of sugar. The increased sugar content in the weed makes it attractive to cattle and sheep, with disastrous results because the toxic level of the plant remains high as the sugar content increases. In Sudan grass 2,4,5-T increased the hydrocyanic acid content by 60 percent, a level in some cases toxic to animals.

Pesticide Resistance in Animals and Plants

The evolutionary impact of pesticides on animals and plants is evidenced by the number of species which have evolved high levels of tolerance to various pesticides. For example, a house mouse population selected for resistance to DDT increased its tolerance to DDT 2-fold in just 10 generations. A pine-mouse population studied in the field was reported to have a 12-fold level of tolerance to endrin above usual levels.

Mosquito fish populations inhabiting streams in the cotton belt evolved significant levels of resistance to DDT, strobane, toxaphene, chlordane, aldrin, heptachlor, dieldrin, endrin, and dursban. Extremely high increases in level of tolerance were reported for strobane (300-fold), endrin (120-fold), toxaphene (40-fold), dieldrin (20-fold), and chlordane (20-fold).

Two frog populations from a cotton-growing region possessed a significant level of resistance to DDT.

As might be expected, insect and mite populations with high levels of resistance to pesticides have been found in many parts of the world. Of nearly 2,000 pest insect and mite species, a total of 225 species has been reported resistant to pesticides. Of these species, 121 species were crop pests, 97 man and animal pests, 6 stored-product pests, and 1 forest pest. In at least one instance the level of resistance had increased 25,000-fold.

Although the evidence is not conclusive, there is strong suggestion of the presence of resistance to 2,4-D in some plants.

Disease Susceptibility

Pesticides caused animals to be more susceptible to certain diseases. For instance, the exposure of mallard ducklings to Aroclor (PCB) increased the susceptibility of the ducklings to duck hepatitus virus. Also, evidence suggests that the exposure of fish to carbaryl and 2,4-D reduced their natural resistance to a microsporidian parasite.

Biological Concentration

The ability of animals and plants to concentrate many types of pesticides in their body tissues appears to be a common physiological phenomenon. The chlorinated insecticides have the greatest affinity for this type of process. The tremendous capacity for concentrating pesticides is best illustrated with oysters and waterfleas. Oysters were able to take DDT at 1 ppb in seawater and concentrate it 70,000 times in their bodies. Waterfleas were even more efficient; they were able to take DDT at 0.5 ppb in water and concentrate it 100,000 times in their bodies.

Normally, the capacity for biological concentration is not as great as this, but when the event is repeated through several links in the food chain, extremely high concentrations of pesticide residues can occur in the species at the top of the food chain.

This happened with the food chain involving soil, earthworms, and robins. Starting with a DDT level in the soil of 9.9 ppm, it reached 141 ppm in the earthworms and 444 ppm in robins. This high concentration in the robins was toxic to some birds.

Persistence

Persistent pesticides have the advantage of remaining in the environment for long periods and thereby being effective in pest control over long periods of time with fewer applications needed. The obvious disadvantage is that the longer the chemical poisons persist, the greater are the chances that they will move out of the treated area via either soil, water, air, or organisms and cause harm to non-target organisms.

Of the insecticides, arsenic at reported use dosages remained in the environment for an estimated 40 years. The chlorinated insecticides also persist for long periods ranging from 6 months to 30 years, depending on the chemical, its dosage, and characteristics of the environment. DDT, for example, at only 1 lb/A persisted in a forest environment for 9 years with little decline in the residue level. Based on the rate of disappearance, the estimation was that DDT even at this low dosage would endure for 30 years. Yet for other economic poisons like malathion, residues may persist for only a few days.

It should be pointed out that even with persistent chlorinated insecticides certain limited amounts can be released into the environment without important ecological effects. This level of release, if known, would help develop rational use programs for pesticides.

Pesticide Movement and Residues in the Environment

The presence of pesticides is generally widespread in the environment, and movement throughout the environment is related in large measure to their persistence. Obviously, the longer a chemical remains in the environment, the greater is the opportunity that it will spread or be transported to another location in the environment.

Antarctic seals and penguins far distant from the application of any pesticides are contaminated with DDT. Also, in the Arctic region seals, caribou, and polar bears contain DDT. A great variety of non-target mammals, birds, fishes, and insects are known to contain residues of numerous kinds of pesticides including the highly toxic mercury compounds. Accumulations of pesticide residues in some resistant non-target species have become sufficiently high to be lethal to some individuals of the species itself and to some predators which feed on them.

Residues of pesticides in soil have been investigated extensively, and persistence in soil was discussed above.

Various pesticide residues are found at low levels in water throughout the United States; however, in the southeast and far west, pesticide residues were present at fairly high levels.

A surprising finding was that pesticides also were detected at fairly high levels in the atmosphere. Insecticide levels in areas far distant from any treated area ranged from below detectable levels to 23 ng/m^3 of DDT, for example. Evidently, movement through the atmosphere provides an excellent means for transport of some pesticides to widely dispersed habitats.

The amount of pesticide released into the atmosphere above the crop, but which does not reach the treated crop, is well illustrated with aircraft spraying. For example, in treating corn by aircraft only about 20 percent of the DDT sprayed from aircraft reached the corn when measured at tassel height.

Pesticides accumulated in living organisms may also travel long distances; the impact of this means of pesticides moving out through the environment probably has been underestimated.

Herbicides are generally less persistent than insecticides, but there are materials like picloram and monuron which will persist for 2 to 5 years in the soil. Fungicides also break down rather rapidly, but some like mercury compounds may leave various stable forms free to move in the biosphere for many years.

Wildlife Management

Although, as documented, pesticides can harm wildlife, with careful use they may also benefit wildlife and therefore be employed in some phases of wildlife management.

Insecticides, for example, are sometimes applied to prevent a serious insect pest from denuding a forest of its leaves and making it an unsuitable habitat for some wildlife species. Herbicides have been employed to alter directly the vegetational types growing in a habitat to improve the habitat for food and shelter for deer, grouse, turkey, and quail.

Herbicides have also been used to control water hyacinth and other aquatic weeds, thereby improving the freshwater habitat for sport fish. The quantity of herbicide used for this purpose alone totals several tons per year.

Conclusions

Available evidence suggests that current methods of pesticide use are a serious hazard to some species which make up our environmental life system. Pesticides, especially the chlorinated insecticides (DDT, dieldrin, toxaphene, chlordane, TDE, aldrin, and heptachlor) already have caused measurable damage to non-target bird, fish, and beneficial insect populations. The prime reason for these chemicals being particularly hazardous is their persistence, movement through the ecosystem, and characteristics for biological concentration in the food chain.

Data on the detrimental effects of other pesticides is spotty, but there is sufficient evidence for concern. More information on the impact these economic poisons are having on non-target species is needed before additional plant and animal species are affected.

Based on available information, some generalizations can be made about the effects of insecticides, herbicides, and fungicides on populations and communities of natural species:

1. Pesticides tend to reduce significantly the numbers of individuals of some species in biotic communities, which has the similar ecological effect of reducing the number of species.
2. An important reduction in the number of species in a community may lead to instability within that community and subsequently to population outbreaks because of alteration in the normal check-balance structure of the community.
3. After pesticide applications the species populations most likely to increase in numbers are those in the lower part of the food chain, that is, the plant feeders. This is, in part, because the parasitic and predaceous enemies which naturally help control numbers of plant feeders often are more susceptible to pesticide pollution effects.
4. In addition, any effective loss of species or intense fluctuations in number of species low in the food chain may adversely affect the dependent predator and parasitic species at the top of the food chain. This in turn further disrupts the structure and ultimately the stability of the natural community.

APPENDICES

Appendix A—Abbreviations Used

ppm, parts per million (parts in 10^6 parts) is the number of parts of toxicant per million parts of the substance in question (not necessarily in solution); these may include residues in soil, water, or whole animals.

ppb, parts per billion (parts in 10^9 parts), is the number of parts of toxicant per billion parts of the substance in question.

mg/kg, milligrams per kilogram, is used to designate the amount of toxicant required per kilogram of body weight of test organisms to produce a designated effect, usually the amount necessary to kill 50 percent of the test animals.

μg, microgram, 1/1,000,000 of gram.

ng, nanogram, 1/1,000,000,000 of a gram.

LD_{50}, median lethal dose, is the milligrams of toxicant per kilogram of body weight lethal to 50 percent of the test animals to which it is administered under the conditions of the experiment.

LC_{50}, median lethal concentration, is the concentration (ppm or ppb) of toxicant in the environment (usually water) which kills 50 percent of the test organisms exposed.

EC_{50}, median effective concentration, is the concentration (ppm or ppb) of toxicant in the environment (usually water) which produces a designated effect in 50 percent of the test organisms exposed.

Appendix B—Common and Scientific Names of Animals and Plants

ANIMALS

Mammals

beaver—*Castor canadensis*
beluga—*Delphinapterus leucas*
black bear—*Euarctos americanus*
buff-bellied chipmunk—*Eutamias amoenus luteiventris*
caribou—*Rangifer tarandus*
coeur d'Alene chipmunk—*Eutamias ruficaudus simulans*
Columbian ground squirrel—*Citellus columbianus*
common seal—*Phoca vitulina*
cotton rat—*Sigmodon hispidus*
cottontail rabbit—*Sylvilagus floridanus*
crab-eater seal—*Lobodon carcinophagus*
domestic goat—*Capra aegagrus*
elk—*Cervus canadensis*
fur seal—*Callorhinus ursinus*
gray whale—*Rhachianectes glaucus*
gray seal—*Halichoerus grypus*
guinea pig—*Cavia porcellus*
harp seal—*Pagophilus groenlandicus*
jumping mouse—*Zapus princeps*
mink—*Mustela vison*
moose—*Rangifer caribou*
mouse (house)—*Mus musculus*
mule deer—*Odocoileus hemionus*
Northern white-footed mouse—*Peromyscus leucopus noveboracensis*
old field mouse—*Peromyscus polionotus*
oogruk seal—*Erignathus barbatus*
pine mouse—*Pitymys pinetorum*
pine squirrel—*Tamiasciurus hudsonicus*
pocket gopher—*Thomomys talpoides*
polar bear—*Thalarctos maritimus*
polecat—*Mustela putorius*
porpoise—*Phocoena phocoena*
prairie deer mouse—*Peromyscus maniculatus bairdii*
prairie vole—*Microtus ochrogaster*
pronghorn antelope—*Antilocapra americana*
rabbit—*Oryctolagus cuniculus*
rat—*Rattus norvegicus*
red-backed mouse—*Clethrionomys gapperi saturatus*
roe deer—*Capreolus capreolus*
sagebrush white-footed mouse—*Peromyscus maniculatus artemisiae*
sage grouse—*Centrocercus urophasianus*
Weddell seal—*Leptonychotes weddelli*
white-footed mouse—*Peromyscus leucopus leucopus*
white-tailed deer—*Odocoileus virginianus*
white-tailed jackrabbit—*Lepus townsendii*
wild hare—*Lepus americanus*

Birds

Adelie penguin—*Pygoscelis adeliae*
American merlin—*Falco columbarius*
American redstart—*Setophaga ruticilla*
American sparrow hawk—*Falco sparverius*
Ashy petrel—*Oceanodroma homochroa*
Atlantic gannet—*Sula bassana*
bald eagle—*Haliaeetus leucocephalus leucocephalus*
barn owl—*Tyto alba*
Bengalese finch—*Lonchura striata*
Bermuda petrel—*Pterodroma cahow*
black duck—*Melanitta nigra*
black tern—*Chlidonias nigra*
blue grouse—*Dendragapus obscurus obscurus*
blue waxbill—*Uraeginthus angolensis angolensis*
blue-winged teal—*Anas discors*
bobwhite quail—*Colinus virginianus*
Brandt's cormorant—*Phalacrocorax penicillatus*
brown pelican—*Pelecanus occidentalis*
bullfinch—*Pyrrhula pyrrhula*
buzzard—*Buteo buteo*
California quail—*Lophortyx californicus*
Canada goose—*Branta canadensis*
carrion crow—*Corvus corone corone*
Cassin's auklet—*Ptychoramphus aleuticus*

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chinstrap penguin—*Pygoscelis antarctica*
chukar partridge—*Alectoris graeca*
common tern—*Sterna hirundo*
coot—*Fulica americana*
coturnix—*Coturnix coturnix japonica*
cowbird—*Molothrus ater*
double-crested cormorant—*Phalacrocorax auritus auritus*
eider duck—*Somateria mollissima*
emperor penguin—*Aptenodytes forsteri*
fork-tailed petrel—*Oceanodroma furcata*
fulmar—*Fulmarus glacialis*
fulvous tree duck—*Dendrocygna bicolor*
golden eagle—*Aquila chrysaetos*
gray partridge—*Perdix perdix*
great blue heron—*Ardea herodias*
great crested grebe—*Podiceps cristatus*
green white-eye—*Zosterops virens*
guillemot—*Uria aalge*
herring gull—*Larus argentatus smithsonianus*
hooded crow—*Corvus corone cornix*
house finch—*Carpodacus mexicanus*
house sparrow—*Passer domesticus*
Japanese stork—*Ciconia ciconia boyciana*
Jardine's babbler—*Turdoides jardinei jardinei*
kestrel—*Falco tinnunculus*
kittiwake—*Rissa tridactyla*
Kurrichane thrush—*Turdus libonyanus libonyanus*
lesser sandhill crane—*Grus canadensis canadensis*
lesser scaup—*Aythya affinis*
little egret—*Egretta garzetta garzetta*
long-eared owl (European)—*Asio otus*
magpie—*Pica pica*
mallard duck—*Anas platyrhynchos*
[illegible] duck—*[illegible]*
merlin—*Falco columbarius*
moorhen—*Gallinula chloropus*
mountain chickadee—*Parus gambeli*
mourning dove—*Zenaidura macroura*
myrtle warbler—*Dendroica coronata*
old-squaw duck—*Clangula hyemalis*
Oregon junco—*Junco hyemalis oreganus*
osprey—*Pandion haliaetus carolinensis*
parula warbler—*Compsothlypis americana*
peregrine falcon—*Falco peregrinus*
prairie chicken—*Tympanuchus cupido*
prairie falcon—*Falco mexicanus*
prairie sharp-tailed grouse—*Pedioecetes phasianellus campestris*
raven—*Corvus corax*
razorbill—*Alca torda*
red-eyed vireo—*Vireo olivaceus*
red-legged partridge—*Alectoris rufa*
red-wing blackbird—*Agelaius phoeniceus*
ring-billed duck—*Aythya collaris*
ring-billed gull—*Larus delawarensis*
ringdove—*Streptopelia risoria*
ring-necked pheasant—*Phasianus colchicus*
robin—*Turdus migratorius*
rook—*Corvus frugilegus*
ruffed grouse—*Bonasa umbellus*
sage grouse—*Centrocercus urophasianus*
shoveler—*Spatula clypeata*
sora—*Porzana carolina*
sparrow hawk (European)—*Accipiter nisus*
speckled coly—*Colius striatus striatus*
tawny owl—*Strix aluco*
tree swallow—*Iridoprocne bicolor*
Western grebe—*Aechmophorus occidentalis*
Western gull—*Larus occidentalis*
white owl—*Aluco pratincola*
white pelican—*Pelecanus erythrorhynchos*
white-tailed eagle—*Haliaeetus albicilla*
white-winged dove—*Zenaida asiatica*
wild turkey—*Meleagris gallopavo*
wood pigeon (English)—*Columba palumbus*
yellow-eye—*Serinus mozambicus mozambicus*

Fishes

alewife—*Pomolobus pseudoharengus*
American smelt—*Osmerus mordax*
anchovy—*Engraulis mordax*
Atlantic croaker—*Micropogon undulatus*
Atlantic salmon—*Salmo salar*
black bullhead—*Ictalurus melas*
bluegill—*Lepomis macrochirus*
bluntnose minnow—*Pimephales notatus*
brill—*Scophthalmus rhombus*
brook trout—*Salvelinus fontinalis*
brown bullhead—*Ictalurus nebulosus*
brown trout—*Salmo trutta*
carp—*Cyprinus carpio*
channel catfish—*Ictalurus punctatus*
chinook salmon—*Oncorhynchus tshawytscha*
chub—*Squalius cephalus*
cod—*Gadus callarias*
coho salmon—*Oncorhynchus kisutch*
cutthroat trout—*Salmo clarkii lewisi*
English sole—*Parophrys vetulus*
fathead minnow—*Pimephales promelas*
golden shiner—*Notemigonus crysoleucas*
goldfish—*Carassius auratus*
green sunfish—*Lepomis cyanellus*
[illegible] fish—*[illegible]*
guppy—*Lebistes reticulatus*
haddock—*Gadus aeglefinus*
hake—*Merluccius productus*
halibut—*Hippoglossus hippoglossus*
harlequin fish—*Rasbora heteromorpha*
herring—*Clupea harengus*
lake chubsucker—*Erimyzon sucetta*
Lake Emerald shiner—*Notropis atherinoides*
lake trout—*Salvelinus namaycush*
landlocked salmon—*Salmo salar sebago*
largemouth bass—*Micropterus salmoides*
longnose killifish—*Fundulus similis*
mackerel—*Scomber scombrus*
mosquito fish—*Gambusia affinis*
[illegible]—*Fundulus [illegible]*
mountain whitefish—*Prosopium williamsoni*
mullet—*Mugil cephalus*
northern puffer—*Sphaeroides maculatus*

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ocean perch—*Sebastodes alutus*
pike—*Esox lucius*
pinfish—*Lagodon rhomboides*
plaice—*Pleuronectes platessa*
pumpkinseed—*Lepomis gibbosus*
rainbow trout—*Salmo gairdneri*
rainwater killifish—*Lucania parva*
redear—*Lepomis microlophus*
redfin shiner—*Notropis umbratilis*
sheepshead—*Cyprinodon variegatus*
shiner perch—*Cymatogaster aggregata*
silver salmon—*Oncorhynchus kisutch*
smallmouth bass—*Micropterus dolomieu*
speckled dace—*Rhinichthys osculus carringtoni*
spot—*Leiostomus xanthurus*
spottail minnow—*Notropis hudsonius*
starry flounder—*Platichthys stellatus*
stickleback—*Gasterosteus aculeatus*
striped bass—*Roccus saxatilis*
striped mullet—*Mugil cephalus*
taillight shiner—*Notropis maculatus*
three-spined stickleback—*Gasterosteus aculeatus*
tidewater silverside—*Menidia beryllina*
tilapia—*Tilapia aurea*
true cod—*Gadus macrocephalus*
turbot—*Scophthalmus maximus*
walleye—*Stizostedion vitreum vitreum*
warasubo—*Odontamblyopus rubicundus*
white catfish—*Ictalurus catus*
white crappie—*Pomoxis annularis*
whitefish—*Coregonus clupeiformis*
white mullet—*Mugil curema*
whiting—*Gadus merlangus*
yellow bullhead—*Ictalurus natalis*
yellow perch—*Perca flavescens*
yellowtail rockfish—*Sebastodes flavidus*

Amphibians

bullfrog—*Rana catesbeiana*
chorus frog—*Pseudacris triseriata*
Fowler's toad—*Bufo woodhousii fowleri*
red-backed salamander—*Plethodon cinereus*

Reptiles

box turtle—*Terrapene c. carolina*
garter snake—*Thamnophis sirtalis*

Molluscs

Asiatic clam—*Corbicula manilensis*
common little neck clam—*Protothaca staminea*
common Washington clam—*Saxidomus nuttalli*
crested oyster—*Ostrea equestris*
eastern oyster—*Crassostrea virginica*
European oyster—*Ostrea edulis*
fresh-water mussel—*Elliptio complanatus*
hooked mussel—*Brachidontes recurvus*
Northern quahog—*Mercenaria mercenaria*
Pacific oyster—*Crassostrea gigas*
soft-shell clam—*Mya arenaria*

Arthropods and Annelids

apple mealy bug—*Phenacoccus aceris*
azuki-bean weevil—*Callosobruchus chinensis*
blue crab—*Callinectes sapidus*
brown shrimp—*Penaeus aztecus*
cabbage aphid—*Brevicoryne brassicae*
codling moth—*Carpocapsa pomonella*
cottony-cushion scale—*Icerya purchasi*
Dungeness crab—*Cancer magister*
European corn borer—*Pyrausta nubilalis*
European red mite—*Panonychus ulmi*
ghost shrimp—*Callianassa affinis*
grass shrimp—*Palaemonetes vulgaris*
gypsy moth—*Porthetria dispar*
hermit crab—*Pagurus longicarpus*
honeybee—*Apis mellifera*
housefly—*Musca domestica*
Japanese beetle—*Popillia japonica*
King crab—*Xiphosurus sowerbyi*
leaf-cutting bee—*Megachile rotundata*
oriental fruit moth—*Grapholitha molesta*
Pacific spider mite—*Tetranychus pacificus*
peach aphid—*Myzus persicae*
pink shrimp—*Penaeus duorarum*
purple urchin—*Strongylocentrotus purpuratus*
range caterpillar—*Hemileuca oliviae*
red-banded leaf roller—*Argyrotaenia velutinana*
red crawfish—*Procambarus clarki*
rice stem-borer—*Chilo plejadellus*
sand shrimp—*Crangon septemspinosa*
shore crab—*Carcinides maenas*
short-spired purple snail—*Thais emarginata*
spruce budworm—*Choristoneura fumiferana*
two-spotted mite—*Tetranychus telarius*
vedalia—*Rhodolia cardinalis*
white shrimp—*Penaeus setiferus*
wireworm—*Ctenicera aeripennis destructor*
yellow scale—*Aonidiella citrina*

PLANTS

American elm—*Ulmus americana*
arrowgrass—*Triglochin maritima*
aspen—*Populus tremuloides*
bitterbrush—*Purshia tridentata*
black cherry—*Prunus serotina*
black oak—*Quercus velutina*
box elder—*Acer negundo*
Canada thistle—*Carduus arvense*
chokecherry—*Prunus virginiana*
Concord grape—*Vitis labruscana*
cottonwood—*Populus deltoides*
creeping thistle—*Cirsium arvense*
downy rabbitbrush—*Chrysothamnus puberulus*

jimson weed—*Datura stramonium*
kale—*Brassica oleracea acephala*
milfoil—*Myriophyllum spicatum*
round-leafed mallow—*Malva neglecta*
Russian pigweed—*Amaranthus retroflexus*
rye—*Secale cereale*
scarlet oak—*Quercus coccinea*
serviceberry—*Amelanchier alnifolia*
silver sagebrush—*Artemisia cana*
snowberry—*Symphoricarpos oreophilus*
snowbrush—*Ceanothus velutinus*
spikerush—*Eleocharis palustris*
Sudan grass—*Sorghum vulgare* var. *sudanense*
sycamore—*Platanus occidentalis*
three square—*Scirpus americanus*
threetip sagebrush—*Artemisia tripartita*
tokay grape—*Vitis vinifera*
velvet-leaf—*Abutilon theophrasti*
white oak—*Quercus alba*
wild parsnip—*Pastinaca sativa*

Appendix C—Index of Pesticides

COMMON AND CHEMICAL NAMES

Page

AAT. See PARATHION.
AAtrex. See ATRAZINE.
ABATE 3
  Chemical name: O,O,O',O'-tetramethyl O,O'-thiodi-p-phenylene phosphorothioate
  Other name: Biothion
  Action: Insecticide
Acaraben. See CHLOROBENZILATE.
Acaralate. See CHLOROPROPYLATE.
Accelerate. See ENDOTHALL.
Accothion. See FENITROTHION.
Acricid. See BINAPACRYL.
ACROLEIN 65
  Chemical name: 2-propenal
  Other names: Acrylaldehyde, Aqualin, Aqualin Biocide, Aqualin Slimicide
  Action: Herbicide
Acrylaldehyde. See ACROLEIN.
Actidione. See CYCLOHEXIMIDE.
Actril. See IOXYNIL.
Afalon. See LINURON.
Aflix. See FORMOTHION.
Afos. See MECARBAM.
Agridip. See COUMAPHOS.
Agroxone. See MCPA.
Akar. See CHLOROBENZILATE.
ALDRIN 3
  Chemical name: 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4-endo-exo-5,8-dimethanonaphthalene
  Other names: Aldrosol, Drinox, HHDN, Octalene, Seedrin
  Action: Insecticide.
Aldrosol. See ALDRIN.
Alkron. See PARATHION.
Alleron. See PARATHION.
ALLETHRIN 6
  Chemical name: *dl*-2-allyl-4-hydroxy-3-methyl-2-cyclopenten-1-one ester of *dl-cis trans*-chrysanthemum monocarboxylic acid
  Other names: Pallethrine, Pynamin
  Action: Insecticide.
Allidochlor. See CDAA.

Page

Allisan. See DICLORAN.
Alltox. See TOXAPHENE.
Ambox. See BINAPACRYL.
Amicide. See AMS.
AMETRYNE 65
  Chemical name: 2-(ethylamino)-4-(isopropylamino)-6-(methylthio)-s-triazine
  Other name: Gesapax
  Action: Herbicide.
AMIBEN 65
  Chemical name: 3-amino-2,5-dichlorobenzoic acid
  Other name: Chloramben
  Action: Herbicide
AMINOCARB 6
  Chemical name: 4-(dimethylamino)-m-tolyl methylcarbamate
  Other name: Matacil
  Action: Insecticide.
α-AMINO-2,6-DICHLORO-BENZALDOXIME 65
  Action: Herbicide.
Aminotriazole. See AMITROLE.
Amitrol T.L. See CYTROL AMITROLE-T
AMITROLE 66
  Chemical name: 3-amino-1,2,4-triazole
  Other names: Aminotriazole, ATA, Weedazol
  Action: Herbicide.
Ammate. See AMS.
AMMONIUM CHLORIDE 157
  Action: Fungicide.
AMMONIUM HYDROXIDE 157
  Action: Fungicide.
AMMONIUM THIOCYANATE 66
  Other name: Amthio
  Action: Herbicide.
AMS 67
  Chemical name: ammonium sulfamate
  Other names: Amicide, Ammate
  Action: Herbicide.
Amthio. See AMMONIUM THIOCYANATE.
Anofex. See DDT.
Anthio. See FORMOTHION.
Anthon. See TRICHLORFON.

Page

Antiphen. See DICHLOROPHEN.
Antracol. See PROPINEB.
Aphamite. See PARATHION.
Aquacide. See DIQUAT.
Aqualin. See ACROLEIN.
Aqualin Biocide. See ACROLEIN.
Aqualin Slimicide. See ACROLEIN.
Aquathol. See ENDOTHALL.
Aracide. See ARAMITE.
ARAMITE 7
  Chemical name: 2(p-tert-butylphenoxy)-1-methylethyl-2'-chloroethyl sulfite
  Other names: Aracide, Niagaramite
  Action: Insecticide.
Arasan. See THIRAM.
Arathane. See DINOCAP.
AROCHLORS 7
  Chemical name: mixture of chlorinated terphenyls
  Other names: Chlorinated biphenyls, PCB's, polychlorinated biphenyls
  Action: Insecticide
Arprocarb. See PROPOXUR.
Aspor. See ZINEB.
ASULAM 87
  Chemical name: methyl 4-aminobenzenesulfonyl carbamate
  Action: Herbicide.
Asuntol. See COUMAPHOS.
ATA. See AMITROLE.
Atlacide. See SODIUM CHLORATE.
Atlas "A". See SODIUM ARSENITE.
Atratol. See SODIUM CHLORATE.
ATRAZINE 87
  Chemical name: 2-chloro-4-ethylamino-6-isopropylamino-s-triazine
  Other names: AAtrex, Fenamine, Fenatrol, Gesaprim, Primatol A
  Action: Herbicide.
Avadex. See DIALLATE.
Avicol. See PCNB.
AZIDE 88
  Action: Herbicide.
Azidithion. See MENAZON.
AZINPHOS-METHYL 8
  Chemical name: *O,O*-dimethyl *S*-(4-oxo-1,2,3-benzotriazin-3(4H)-ylmethyl)phosphorodithioate
  Other names: Carfene, DBD, Gusathion, Gusathion M, Gustathion, Guthion, Methyl Guthion
  Action: Insecticide.
Azodrin. See MONOCROTOPHOS.
Balan. See BENEFIN.
Balfin. See BENEFIN.
Banex. See DICAMBA.
Bantrol. See IOXYNIL.
Banvel D. See DICAMBA.
Barbamate. See BARBAN.
BARBAN 88
  Chemical name: 4-chloro-2-butynyl m-chlorocarbanilate
  Other names: Barbamate, Carbyne
  Action: Herbicide.

Page

Basic copper chloride. See COPPER OXYCHLORIDE.
Basudin. See DIAZINON.
Baygon. See PROPOXUR.
Baytex. See FENTHION.
BENAZOLIN 88
  Chemical name: 4-chloro-2-oxobenzothiazolin-3-ylacetic acid
  Other names: Cornox CWK, Legumex Extra, LeyCornox
  Action: Herbicide.
BENEFIN 88
  Chemical name: *N*-butyl-*N*-ethyl-α,α,α-trifluoro-2,6-dinitro-p-toluidine
  Other names: Balan, Balfin, Binnell, Quilan
  Action: Herbicide.
Benzac. See 2,3,6-TBA.
BENZENETHIOL 137
  Action: Fungicide.
BENZOIC ACID 137
  Action: Fungicide.
BHC. See LINDANE.
Bichloride of mercury. See CORROSIVE SUBLIMATE.
Bidrin. See DICROTOPHOS.
BINAPACRYL 9
  Chemical name: 2 sec-butyl-4,6-dinitrophenyl-3-methyl-2-butenoate
  Other names: Acricid, Ambox, Dinoseb methacrylate, Endosan, Morocide
  Action: Insecticide.
Binnell. See BENEFIN.
Bioquin. See COPPER-8-QUINOLINOLATE.
Biostat PA. See OXYTETRACYCLINE.
Biothion. See ABATE.
Birlane. See CHLORFENVINPHOS.
BIUREA 137
  Action: Fungicide.
Bladan. See TEPP.
Blattanex. See PROPOXUR.
BMM. See UREABOR.
Borascu. See BORAX.
BORAX 89
  Chemical name: sodium tetraborate decahydrate
  Other names: Borascu, Gerstley Borate, Polybor, Tronabor
  Action: Herbicide.
Botran. See DICLORAN.
Botrilex. See PCNB.
Brassicol. See PCNB.
Brestan. See FENTIN ACETATE.
Brimstone. See SULFUR.
Brofene. See BROMOPHOS.
Brominal. See BROMOXYNIL.
BROMOPHOS 9
  Chemical name: *O*-(4-bromo-2,5-dichlorophenyl)*O,O*-dimethylphosphorothioate
  Other names: Brofene, Nexion
  Action: Insecticide.
BROMOXYNIL 89
  Chemical name: 3,5-dibromo-4-hydroxybenzonitrile
  Other name: Brominal
  Action: Herbicide.

Page
BUSAN 137
Action: Fungicide.
Butacide. See PIPERONYL BUTOXIDE.
Butter of Zinc. See ZINC CHLORIDE.
CACODYLIC ACID 89
Chemical name: dimethylarsinic acid
Other name: Silvisar 510
Action: Herbicide.
Cadminate. See CADMIUM SUCCINATE.
CADMIUM SUCCINATE 138
Chemical name: 60% cadmium succinate (29% metallic basis)
Other name: Cadminate
Action: Fungicide.
CAFFEINE 138
Action: Fungicide.
Caparol. See PROMETRYNE.
CAPTAFOL 138
Chemical name: cis-N[(1,1,2,2-tetrachloroethyl) thio]-4-cyclohexene-1,2-dicarboximide
Other names: Difolatan, Folcid
Action: Fungicide.
CAPTAN 138
Chemical name: N-[(trichloromethyl)thio]-4-cyclohexene-1,2-dicarboximide
Other name: Orthocide 406
Action: Fungicide.
CARBARYL 9
Chemical name: 1-naphthyl methylcarbamate
Other name: Sevin
Action: Insecticide.
Carbicron. See DICROTOPHOS.
Carbofos. See MALATHION.
CARBOFURAN 11
Chemical name: 2,3-dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate
Other name: Furadan
Action: Insecticide.
CARBOLIC ACID 139
Chemical name: phenol
Other name: Phenol
Action: Fungicide.
CARBOPHENOTHION 12
Chemical name: S-[(p-chlorophenyl)thiomethyl] O,O-diethyl phosphorodithioate
Other names: Dagadip, Garrathion, Trithion
Action: Insecticide.
Carbophos. See MALATHION.
Carbyne. See BARBAN.
Carfene. See AZINPHOS-METHYL.
Carpene. See DODINE.
Casoron. See DICHLOBENIL.
CDAA 90
Chemical name: 2-chloro-N,N-diallyl acetamide
Other names: Allidochlor, Randox
Action: Herbicide.
CDEC 90
Chemical name: 2-chloroallyl-N,N-diethyldithiocarbamate
Other names: Sulfallate, Vegadex
Action: Herbicide.
Page
Certrol. See IOXYNIL.
Chem Bam. See NABAM.
Chem-Hoe. See PROPHAM.
Chemox General. See DNBP.
Chemox P.E. See DNBP.
Chem Pels C. See SODIUM ARSENITE.
Chem Zineb. See ZINEB.
Chinomethionate. See OXYTHIOQUINOX.
Chiptox. See MCPA.
Chloramben. See AMIBEN.
CHLORANIL 139
Chemical name: 2,3,5,6-tetrachloro-1,4-benzoquinone; also tetrachloro-p-benzoquinone
Other name: Spergon
Action: Fungicide.
Chlorax. See SODIUM CHLORATE.
CHLORBENSIDE 12
Chemical name: p-chlorobenzyl p-chlorophenyl sulfide
Other names: Chlorocide, Chlorparacide, Chlorsulphacide, Midox, Mitox
Action: Insecticide.
Chlordan. See CHLORDANE.
CHLORDANE 12
Chemical name: 1,2,4,5,6,7,8,8-octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene
Other names: Chlordan, Chlor Kil, Corodane, Kypchlor, Octachlor, Octa-Klor, Ortho-Klor, Synklor, Topiclor 20, Velsicol 1068
Action: Insecticide.
CHLORDECONE 14
Chemical name: decachloro-octahydro-1,3,4-metheno-2H-cyclobuta(cd)pentalen-2-one
Other name: Kepone
Action: Insecticide.
CHLOREA 91
Chemical name: formulation with sodium chlorate, sodium metaborate, and monuron
Action: Herbicide.
Chlorfenidim. See MONURON.
Chlorfenson. See OVEX.
CHLORFENVINPHOS 14
Chemical name: 2-chloro-1-(2,4-dichlorophenyl)-vinyl diethyl phosphate
Other names: Birlane, Sapecron, Supona
Action: Insecticide.
CHLORFLURAZONE 91
Chemical name: 4,5-dichloro-2-trifluoromethylbenzimidazole
Action: Herbicide.
Chlorinated biphenyls. See AROCHLORS.
Chlorinated camphene. See TOXAPHENE.
CHLORINE 140
Action: Fungicide.
Chlor Kil. See CHLORDANE.
CHLOROBENZILATE 14
Chemical name: ethyl 4,4'-dichlorobenzilate
Other names: Acaraben, Akar, Folbex, Kop-Mite
Action: Insecticide.
Chlorocide. See CHLORBENSIDE.
Chlorofenizon. See OVEX.

Page
Chlorofos. See TRICHLORFON.
Chloro IPC. See CHLORPROPHAM.
CHLORONITROPROPANE 140
  Chemical name: 1-chloro-1-nitropropane
  Other names: Lanstan, Korax
  Action: Fungicide.
Chlorophenothane. See DDT.
Chloropropham. See CHLORPROPHAM.
CHLOROPROPYLATE 14
  Chemical name: isopropyl 4,4'-dichlorobenzilate
  Other names: Acaralate, Rospin
  Action: Insecticide.
CHLOROTHION 14
  Chemical name: O,O-dimethyl O-(3-chloro-4-nitrophenyl) phosphorothioate
  Other name: Chlorthion
  Action: Insecticide.
Chloroxone. See 2,4-D.
CHLOROXURON 91
  Chemical name: 3-[p-(p-chlorophenoxy) phenyl]-1,1-dimethylurea
  Other name: Tenoran
  Action: Herbicide.
Chlorparacide. See CHLORBENSIDE.
CHLORPROPHAM 91
  Chemical name: isopropyl m-chlorocarbanilate
  Other names: Chloro IPC, Chloropropham, CICP, CIPC, Furloe, Sprout Nip, Y-3
  Action: Herbicide.
Chlorsulphacide. See CHLORBENSIDE.
Chlorthal-methyl. See DCPA.
CHLORTHIAMID 92
  Chemical name: 2,6-dichlorothiobenzamide
  Other name: Prefix
  Action: Herbicide.
Chlorthiepin. See ENDOSULFAN.
Chlorthion. See CHLOROTHION.
Cinerins. See PYRETHRINS.
CIODRIN 15
  Chemical name: α-methylbenzyl-3-hydroxy-cis-crotonate dimethylphosphate
  Action: Insecticide.
CIPC. See CHLORPROPHAM.
Colloidox. See COPPER OXYCHLORIDE.
COPPER CARBONATE 140
  Other name: Malachite
  Action: Fungicide.
Copper oxinate. See COPPER 8-QUINOLINOLATE.
COPPER OXYCHLORIDE 140
  Other names: Basic copper chloride, Colloidox, Coprantol, Cosyan, Cupramar, Cupravit, Cupros, Vitriuivre
  Action: Fungicide.
COPPER 8-QUINOLINOLATE 140
  Other names: Bioquin, Copper oxinate, Cunilate 2472, Oxine-copper
  Action: Fungicide
COPPER SULFATE 140
  Action: Fungicide.
Coprantol. See COPPER OXYCHLORIDE.
Co-Ral. See COUMAPHOS.

Page
Cornox CWK. See BENAZOLIN.
Cornox RK. See DICHLORPROP.
Corodane. See CHLORDANE.
Corothion. See PARATHION.
CORROSIVE SUBLIMATE 142
  Chemical name: mercuric chloride
  Other names: Bichloride of mercury, Fungchex
  Action: Fungicide, Insecticide.
Cotoran. See FLUOMETURON.
COUMAPHOS 15
  Chemical name: O,O-diethyl O-(3-chloro-4-methyl-2-oxo-(2H)-benzopyran-7-yl) phosphorothioate
  Other names: Agridip, Asuntol, Co-Ral, Muscatox, Resitox
  Action: Insecticide.
Cosyan. See COPPER OXYCHLORIDE.
4-CPA 93
  Chemical name: parachlorophenoxyacetic acid
  Other name: Tomatotone
  Action: Herbicide.
CPCBS. See OVEX.
CRESOL 142
  Chemical name: methyl phenol
  Action: Fungicide, Insecticide.
Crop Rider. See 2,4-D.
CRUFOMATE 15
  Chemical name: O-4-tert-butyl-2-chlorophenyl O-methyl methylphosphoramidate
  Other name: Ruelene
  Action: Insecticide.
CRYOLITE 15
  Chemical name: sodium fluoaluminate or sodium aluminofluoride
  Other name: Kryocide
  Action: Insecticide.
Cuman. See ZIRAM.
Cunilate 2472. See COPPER 8-QUINOLINOLATE.
Cupramar. See COPPER OXYCHLORIDE.
Cupravit. See COPPER OXYCHLORIDE.
Cupros. See COPPER OXYCHLORIDE.
Curitan. See DODINE.
CYANO(METHYLMERCURI)GUANIDINE 142
  Chemical name: N-cyano-N'-(methylmercury) guanidine; also methylmercury dicyandiamide
  Other names: Methylmercuric cyanoguanidine, Morsodren, Panodrin A-13, Panogen
  Action: Fungicide.
Cyclodan. See ENDOSULFAN.
CYCLOHEXIMIDE 142
  Chemical name: 3-[2-(3,5-dimethyl-2-oxocyclohexyl)-2-hydroxyethyl]glutarimide
  Other names: Actidione, Naramycin A
  Action: Fungicide.
CYCLOPENTADIENE 142
  Action: Fungicide.
Cygon. See DIMETHOATE.
Cyprex. See DODINE.
Cythion. See MALATHION.

Page

CYTROL AMITROLE-T 99
  Chemical name: mixture of aminotriazole and ammonium thiocyanate
  Other name: Amitrol T.L.
  Action: Herbicide.
2,4-D 99
  Chemical name: 2,4-dichlorophenoxyacetic acid or its sodium salt or amine
  Other names: Chloroxone, Crop Rider, Ded-Weed, Weed-Ag-Bar, Weedar 64, Weed-B-Gon, Weedone
  Action: Herbicide
Dacthal. See **DCPA**.
Dagadip. See **CARBOPHENOTHION**.
DALAPON 100
  Chemical name: 2,2-dichloropropionic acid
  Other names: Ded-Weed, Dowpon, Gramevin, Radapon, Unipon
  Action: Herbicide.
Daphene. See **DIMETHOATE**.
Dasanit. See **FENSULFOTHION**.
Dasinit. See **FENSULFOTHION**.
DATC. See **DIALLATE**.
Dazomet. See **DMTT**.
Dazzel. See **DIAZINON**.
DBCP. See **DIBROMOCHLOROPROPANE**.
DBD. See **AZINPHOS-METHYL**.
2,6-DBN. See **DICHLOBENIL**.
DCMU. See **DIURON**.
DCNA. See **DICLORAN**.
DCPA 101
  Chemical name: dimethyl tetrachloroterephthalate
  Other names: Chlorthal-methyl, Dacthal
  Action: Herbicide.
DDD. See **TDE**.
DDT 16
  Chemical name: dichloro diphenyl trichloroethane
  Other names: Anofex, Chlorophenothane, Dedelo, Genitox, Gesapon, Gesarex, Gesarol, Gyron, Ixodex, Kopsol, Neocid, Pentachlorin, Rukseam, Zerdane
  Action: Insecticide.
DDVF. See **DICHLORVOS**.
DDVP. See **DICHLORVOS**.
Dechlorane. See **MIREX**.
Dedelo. See **DDT**.
Dedevap. See **DICHLORVOS**.
Ded-Weed. See **2,4-D, DALAPON**.
Ded-Weed Brush Killer. See **2,4,5-T**.
DEF 102
  Chemical name: *S,S,S*-tributyl phosphorotrithioate
  Other names: De-Green, E-Z-Off D, Fos Fall "A," Ortho phosphate defoliant
  Action: Herbicide.
De-Fend. See **DIMETHOATE**.
De-Fol-Ate. See **SODIUM CHLORATE**.
De-Green. See **DEF**.
Delan. See **DITHIANON**.

Page

Deleaf Defoliant. See **MERPHOS**.
DELMETON 26
  Action: Insecticide.
Delnav. See **DIOXATHION**.
DELRAD 143
  Chemical name: dehydro abietylamine acetate, tetrahydro abietylamine acetate, dihydro abietylamine acetate
  Action: Fungicide.
DEMETON 20
  Chemical name: O,O-diethyl O(and S)-2-(ethylthio)ethyl phosphorothioates
  Other names: Demeton O, Mercaptophos, Systox
  Action: Insecticide.
DEMETON METHYL 28
  Chemical name: O,O-dimethyl O(and S)-2(ethylthio)ethyl phosphorothioates
  Other names: Demeton-S-methyl, Meta-Systox, Methyl demeton, Methylsystox
  Action: Insecticide.
Demeton *O*. See **DEMETON**.
Demeton-S-methyl. See **DEMETON METHYL**.
Derris. See **ROTENONE**.
Des-i-cate. See **ENDOTHALL**.
2,4-DES Na. See **SESONE**.
DEXON 143
  Chemical name: p-dimethylaminobenzenediazo sodium sulfonate
  Action: Fungicide.
Dextrone. See **DIQUAT**.
DIALLATE 102
  Chemical name: *S*-2,3-dichloroallyl diisopropylthiocarbamate
  Other names: Avadex, DATC
  Action: Herbicide.
Dianisyltrichloroethane. See **METHOXYCHLOR**.
Diazajet. See **DIAZINON**.
Diazide. See **DIAZINON**.
DIAZINON 29
  Chemical name: *O,O*-diethyl *O*-(2-isopropyl-6-methyl-4-pyrimidinyl) phosphorothioate
  Other names: Basudin, Dazzel, Diazajet, Diazide, Gardentox, Spectracide
  Action: Insecticide.
Dibrom. See **NALED**.
DIBROMOCHLOROPROPANE 30
  Chemical name: 1,2-dibromo-3-chloropropane
  Other names: DBCP, Fumazone, Nemafume, Nemagon
  Action: Insecticide.
DICAMBA 103
  Chemical name: 3,6-dichloro-o-anisic acid
  Other names: Banex, Banvel D, Mediben
  Action: Herbicide.
Di-Captan. See **DICAPTHON**.
DICAPTHON 31
  Chemical name: *O*-(2-chloro-4-nitrophenyl)*O,O*-dimethyl phosphorothioate
  Other name: Di-Captan
  Action: Insecticide.

Page

DICHLOBENIL 103
Chemical name: 2,6-dichlorobenzonitrile
Other names: Casoron, Du-Sprex, 2,6-DBN
Action: Herbicide.
DICHLOFENTHION 31
Chemical name: *O*-2,4-dichlorophenyl *O,O*-diethyl phosphorothioate
Other names: Hex-Nema, Tri-VC 13, VC-13 Nemacide
Action: Insecticide.
DICHLOFLUANID 143
Chemical name: *N'*-dichlorofluoromethylthio-*N,N*-dimethyl-*N'*-phenylsulfamide
Other names: Elvaron, Euparen, Euparene
Action: Fungicide.
DICHLONE 143
Chemical name: 2,3-dichloro-1,4-naphthoquinone
Other name: Phygon
Action: Fungicide.
Dichloran. See **DICLORAN**.
DICHLOROPHEN 144
Chemical name: di-(5-chloro-2-hydroxyphenyl) methane
Other names: Antiphen, Preventol
Action: Fungicide.
Dichlorphos. See **DICHLORVOS**.
DICHLORPROP 104
Chemical name: 2-(2,4-dichlorophenoxy)propionic acid
Other names: Cornox RK, 2,4-DP, Hedonal DP, Kildip
Action: Herbicide.
DICHLORVOS 31
Chemical name: 2,2-dichlorovinyl *O,O*-dimethyl phosphate
Other names: DDVF, DDVP, Dedevap, Dichlorphos, Herkol, Mafu, Marvex, Nogos, No-Pest, Nuvan, Oko, Phosvit, Vapona
Action: Insecticide.
DICLORAN 144
Chemical name: 2,6-dichloro-4-nitroaniline
Other names: Allisan, Botran, DCNA, Dichloran, Ditranil
Action: Fungicide.
DICOFOL 32
Chemical name: 4,4'-dichloro-α-(trichloromethyl) benzhydrol
Other name: Kelthane
Action: Insecticide.
DICROTOPHOS 32
Chemical name: 3-hydroxy-*N,N*-dimethyl-cis-crotonamide dimethyl phosphate
Other names: Bidrin, Carbicron, Ektafos
Action: Insecticide.
DIELDRIN 33
Chemical name: 1,2,3,4,10,10-hexachloro-exo-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo-exo-5,8-dimethanonaphthalene, and related compounds
Other names: HEOD, Octalox, Panoram D-31
Action: Insecticide.
Diethyl ethylthioethyl dithiophosphate. See **DISULFOTON**.

Page

Difenson. See **OVEX**.
Difolatan. See **CAPTAFOL**.
DILAN 37
Chemical name: mixture of one part 1,1-bis(p-chlorophenyl)-2-nitropropane and two parts 1,1-bis(p-chlorophenyl)-2-nitrobutane
Other name: Prolan-Bulan Mixture
Action: Insecticide.
DIMANIN 37
Action: Insecticide.
Dimecron. See **PHOSPHAMIDON**.
DIMETHOATE 37
Chemical name: *O,O*-dimethyl *S*-(*N*-methylcarbamoylmethyl) phosphorodithioate
Other names: Cygon, Daphene, De-Fend, Fostion MM, Le-Kit, Perfekthion, Rogor, Roxion, Trimetion
Action: Insecticide.
Dimethoxy-DT. See **METHOXYCHLOR**.
DIMETHRIN 38
Chemical name: 2,4-dimethylbenzyl-2,2-dimethyl-3-(2-methylpropenyl) cyclopropanecarboxylate
Action: Insecticide.
Dimicron. See **PHOSPHAMIDON**.
Dinex. See **DN-111**.
DINOCAP 145
Chemical name: 2-(1-methyl-n-heptyl)-4,6-dinitrophenyl crotonate, with its isomer 4-(1-methyl-n-heptyl)-2,6-dinitrophenyl crotonate
Other names: Arathane, Iscothane, Karathane, Mildex
Action: Fungicide.
Dinoseb. See **DNBP**.
Dinoseb methacrylate. See **BINAPACRYL**.
DIOTHYL 38
Chemical name: O,O-diethyl-O-(2-dimethylamino-4-(methyl-pyrimidin-6-yl))phosphorothioate
Other name: Pyrimithate
Action: Insecticide.
DIOXATHION 38
Chemical name: 2,3-p-dioxanedithiol *S,S*-bis(*O,O*-diethyl phosphorodithioate)
Other names: Delnav, Navadel, Ruphos
Action: Insecticide.
Dipterex. See **TRICHLORFON**.
DIQUAT 104
Chemical name: 1,1'-ethylene-2,2'-dipyridylium dibromide
Other names: Aquacide, Dextrone, FB/2, Reglone
Action: Herbicide.
DISULFOTON 39
Chemical name: *O,O*-diethyl *S*-2-(ethylthio)ethyl phosphorodithioate
Other names: Diethylethylthioethyl dithiophosphate, Di-syston, Dithiodemeton, Dithiosystox, Frumin Al, Frumin G, Solvirex, Thiodemeton
Action: Insecticide.
Disul-Na. See **SESONE**.

Page
Di-syston. See DISULFOTON.
Dithane A-40. See NABAM.
Dithane D-14. See NABAM.
Dithane Z-78. See ZINEB.
DITHIANON 145
  Chemical name: 5,10-dihydroxy-5,10-dioxo naphtho-(2,3b)-p-dithiin-2,3-dicarbonitrile
  Other name: Delan
  Action: Fungicide.
Dithiodemeton. See DISULFOTON.
Dithiosystox. See DISULFOTON.
Ditranil. See DICLORAN.
DIURON 105
  Chemical name: 3-(3,4-dichlorophenyl)-1,1-dimethylurea
  Other names: DCMU, DMU, Karmex, Marmer
  Action: Herbicide.
DMDT. See METHOXYCHLOR.
DMPA 107
  Chemical name: O-(2,4-dichlorophenyl)O'-methyl *N*-isopropylphosphoroamidothioate
  Other name: Zytron
  Action: Herbicide.
DMTP. See FENTHION.
DMTT 145
  Chemical name: tetrahydro-3,5-dimethyl-2H-thiadiazine-2-thione
  Other names: Dazomet, Micofume, Mylone, Preservit
  Action: Fungicide.
DMU. See DIURON.
DN-111 59
  Chemical name: 2-cyclohexyl-4,6-dinitro phenol, dicyclohexylamine salt
  Other names: Dinex, DNOCHP
  Action: Insecticide.
DN-289. See DNBP.
DNBP 107
  Chemical name: 2,4-dinitro-6-sec-butylphenol
  Other names: Chemox General, Chemox P.E., Dinoseb, DN-289, DNOSBP, Dow General, Elgetol 318, Kiloseb, Nitropone C, Premerge, Sinox General
  Action: Herbicide, Insecticide.
DNC. See DNOC.
DNOC 59
  Chemical name: 4,6-dinitro-o-cresol
  Other names: DNC, Elgetol 30, Nitrador, Sinox, Trifocide
  Action: Insecticide, Herbicide, Fungicide.
DNOCHP. See DN-111.
DNOSBP. See DNBP.
DNTP. See PARATHION.
DODINE 145
  Chemical name: n-dodecylguanidine acetate
  Other names: Carpene, Curitan, Cyprex, Doguadine, Melprex, Tridodine
  Action: Fungicide.
Doguadine. See DODINE.
Dowacide G. See SODIUM PENTACHLOROPHENATE.
Dow General. See DNBP.
Dowpon. See DALAPON.
2,4-DP. See DICHLORPROP.
Drinox. See ALDRIN.
Drinox H-34. See HEPTACHLOR.
Drop Leaf. See SODIUM CHLORATE.
DSE. See NABAM.
Duphar. See TETRADIFON.
DURSBAN 40
  Chemical name: O,O-diethyl O-3,5,6-trichloro-2-pyridyl phosphorothioate
  Action: Insecticide.
Du-Sprex. See DICHLOBENIL.
Du-Ter. See FENTIN HYDROXIDE.
Dybar. See FENURON.
Dylox. See TRICHLORFON.
DYRENE 146
  Chemical name: 2,4-dichloro-6-(o-chloroanilino)-s-triazine
  Other names: Kemate, Triasyn
  Action: Fungicide.
Easy-Off D. See MERPHOS.
EC-90 146
  Action: Fungicide.
Ekatin M. See MORPHOTHION.
Ektafos. See DICROTOPHOS.
Elancolan. See TRIFLURALIN.
Elgetol 30. See DNOC.
Elgetol 318. See DNBP.
Elvaron. See DICHLOFLUANID.
Emerald green. See PARIS GREEN.
Emmatos. See MALATHION.
Endosan. See BINAPACRYL.
ENDOSULFAN 41
  Chemical name: 6,7,8,9,10,10-hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzodioxathiepin 3-oxide
  Other names: Chlorthiepin, Cyclodan, Insectophene, Kop-Thiodan, Malix, Malix, Thifor, Thimul, Thiodan
  Action: Insecticide.
Endothal. See ENDOTHALL.
ENDOTHALL 108
  Chemical name: 7-oxabicyclo (2,2,1)heptane-2,3-dicarboxylic acid
  Other names: Accelerate, Aquathol, Des-i-cate, Endothal, Hydrothol, Niagrathal, Tri-Endothal
  Action: Herbicide
ENDRIN 42
  Chemical name: 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo-endo-5,8-dimethanonaphthalene
  Other names: Hexadrin, Mendrin
  Action: Insecticide.
Entex. See FENTHION.
EPH 44
  Action: Insecticide.
Ephirsulphonate. See OVEX.
EPN 44
  Chemical name: O-ethyl O-p-nitrophenyl phenylphosphonothioate, or ethyl p-nitrophenyl thionobenzenephosphonate
  Action: Insecticide.

Page
Eptam. See EPTC.
EPTC 109
Chemical name: S-ethyl-N,N-dipropylthiocarbamate
Other name: Eptam
Action: Herbicide.
Esteron. See SILVEX.
Esteron 245 Concentrate. See 2,4,5-T.
Estonmite. See OVEX.
ETHION
Chemical name: bis(S-diethoxyphosphinothioyl) mercaptomethane 45
Other name: Nialate
Action: Insecticide.
Etisul. See METIRAM.
Ethyl parathion. See PARATHION.
Etilon. See PARATHION.
Etrolene. See RONNEL.
Euparen. See DICHLOFLUANID.
Euparene. See DICHLOFLUANID.
E-Z-Off D. See DEF.
Fall. See SODIUM CHLORATE.
FB/2. See DIQUAT.
FENAC 109
Chemical name: Sodium 2,3,6-trichlorophenylacetate
Other names: Tri-Fen, Tri-Fene
Action: Herbicide.
Fen-All. See 2,3,6-TBA.
Fenamine. See ATRAZINE.
Fenatrol. See ATRAZINE.
Fence Rider. See 2,4,5-T.
Fenchlorfos. See RONNEL.
Fenidim. See FENURON.
FENITROTHION 45
Chemical name: O,O-dimethyl O-(4-nitro-m-tolyl) phosphorothioate
Other names: Accothion, Folithion, MEP, Nuvanol, Sumithion, Sumitomo
Action: Insecticide.
Fenoprop. See SILVEX.
FENSULFOTHION 46
Chemical name: O,O-diethyl O-p-(methylsulfinyl) phenyl phosphorothioate
Other names: Dasanit, Dasinit, Terracur
Action: Insecticide.
FENTHION 46
Chemical name: O,O-dimethyl O[4-(methylthio)-m-tolyl] phosphorothioate
Other names: Baytex, DMPT, Entex, Lebaycid, Mercaptophos, Queletox, Quelatox, Tiguvon
Action: Insecticide.
FENTIN ACETATE 146
Chemical name: triphenyltin acetate
Other name: Brestan
Action: Fungicide.
FENTIN HYDROXIDE 146
Chemical name: triphenyltin hydroxide
Other name: Du-Ter
Action: Fungicide.

Page
Fenulon. See FENURON.
FENURON 110
Chemical name: 3-phenyl-1,1-dimethylurea
Other names: Dybar, Fenidim, Fenulon, PDU
Action: Herbicide.
FERBAM 146
Chemical name: ferric dimethyl dithiocarbamate
Other names: Ferberk, Fermate, Hexaferb, Tricarbamix, Trifungol
Action: Fungicide.
Ferberk. See FERBAM.
Fermate. See FERBAM.
Fernide 850. See THIRAM.
FLUOMETURON 111
Chemical name: 3-(m-trifluoromethylphenyl)-1,1-dimethylurea
Other name: Cotoran
Action: Herbicide.
Folbex. See CHLOROBENZILATE.
Folcid. See CAPTAFOL.
Folex. See MERPHOS.
Folidol. See PARATHION.
Folosan. See PCNB.
Folithion. See FENITROTHION.
FOLPET 147
Chemical name: N-(trichloromethylthio)phthalimide
Other names: Phaltan, Thiophal, Trichloromethylthiophthalimide
Action: Fungicide.
Formaldehyde. See FORMALIN.
FORMALIN 148
Chemical name: methanal
Other name: Formaldehyde
Action: Fungicide.
FORMOTHION 47
Chemical name: O,O-dimethyl S-(N-formyl-N-methyl-carbamoylmethyl)phosphorodithioate
Other names: Aflix, Anthio
Action: Insecticide.
Forstan. See OXYTHIOQUINOX.
Fos-Fall "A." See DEF.
Fostion MM. See DIMETHOATE.
French green. See PARIS GREEN.
Frumin Al. See DISULFOTON.
Frumin G. See DISULFOTON.
Fuklasin. See ZIRAM.
Fumazone. See DIBROMOCHLOROPROPANE.
Fungchex. See CORROSIVE SUBLIMATE.
Furadan. See CARBOFURAN.
α-FURALDEHYDE 148
Action: Fungicide.
Furloe. See CHLORPROPHAM.
Gallotox. See PMA.
Gamaphex. See LINDANE.
Gamma BHC. See LINDANE.
Gammalin. See LINDANE.
Gammex. See LINDANE.
Gammexane. See LINDANE.
Gardentox. See DIAZINON.

Page
GARDONA ... 47
  Chemical name: 2-chloro-1-(2,4,5-trichlorophenyl)vinyl dimethylphosphate
  Other name: Rabon
  Action: Insecticide.
Garlon. See SILVEX.
Garrathion. See CARBOPHENOTHION.
Genitox. See DDT.
Gerstley Borate. See BORAX.
Gesafram. See PROMETONE.
Gesagard. See PROMETRYNE.
Gesamil. See PROPAZINE.
Gesapax. See AMETRYNE.
Gesapon. See DDT.
Gesaprim. See ATRAZINE.
Gesarex. See DDT.
Gesarol. See DDT.
Gesatop. See SIMAZINE.
GLYODIN ... 148
  Chemical name: 2-heptadecylimidazoline acetate
  Action: Fungicide.
Gramevin. See DALAPON.
Gramoxone. See PARAQUAT.
Granosan. See MERCURY.
GRISEOFULVIN ... 148
  Chemical name: 7-chloro-4,6-dimethoxycoumaran-3-one-2-spiro-1'-(2'-methoxy-6'-methylcyclohex-2'-en-4'-one)
  Action: Fungicide.
Gusathion. See AZINPHOS-METHYL.
Gusathion M. See AZINPHOS-METHYL.
Gustathion. See AZINPHOS-METHYL.
Guthion. See AZINPHOS-METHYL.
Gypsine. See LEAD ARSENATE.
Gyron. See DDT.
Hedonal DP. See DICHLORPROP.
HEOD. See DIELDRIN.
HEPTACHLOR ... 47
  Chemical name: 1,4,5,6,7,8,8-heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindane
  Other names: Drinox H-34, Heptamul
  Action: Insecticide.
Heptamul. See HEPTACHLOR.
Herkol. See DICHLORVOS
HETP. See TEPP.
HEXACHLOROPHENE ... 148
  Chemical name: 2,2'-methylene bis(3,4,6-trichlorophenol)
  Other name: Nabac
  Action: Fungicide.
Hexadrin. See ENDRIN.
Hexaferb. See FERBAM.
Hexathane. See ZINEB.
Hexathir. See THIRAM.
Hexazir. See ZIRAM.
Hex-nema. See DICHLOFENTHION.
HHDN. See ALDRIN.
HIPPURIC ACID ... 148
  Action: Fungicide.
Hong Nien. See PMA.
Hormotuho. See MCPA

Page
Hydram. See MOLINATE.
Hydrothol. See ENDOTHALL.
HYDROXYMERCURICHLOROPHENOLS ... 149
  Other names: Semesan, Tersan
  Action: Fungicide.
IPC. See PROPHAM.
Insectophene. See ENDOSULFAN.
Inverton 245. See 2,4,5-T.
IOXYNIL ... 111
  Chemical name: 4-hydroxy-3,5-diiodobenzonitrile
  Other names: Actril, Bantrol, Certrol
  Action: Herbicide.
IPC. See PROPHAM.
Isothane. See DINOCAP.
ISODRIN ... 50
  Chemical name: 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4,5,8-endo-endo-dimethanonaphthalene
  Action: Insecticide.
Isotox. See LINDANE.
Ixodex. See DDT.
Jasmolins. See PYRETHRINS.
Karathane. See DINOCAP.
Karbofos. See MALATHION.
Karmex. See DIURON.
Kelthane. See DICOFOL.
Kemate. See DYRENE.
Kepone. See CHLORDECONE.
Kildip. See DICHLORPROP.
Kilmite 40. See TEPP.
Kiloseb. See DNBP.
Kilsem. See MCPA.
Kilval. See VAMIDOTHION.
Kloben. See NEBURON.
KMH. See MALEIC HYDRAZIDE.
Kop-Mite. See CHLOROBENZILATE.
Kopsol. See DDT.
Kop-Thiodan. See ENDOSULFAN.
Kop-Thion. See MALATHION.
Korax. See CHLORONITROPROPANE.
Korlan. See RONNEL.
Kryocide. See CRYOLITE.
Kuron. See SILVEX.
Kurosal. See SILVEX.
Kypchlor. See CHLORDANE.
Kypfos. See MALATHION.
Kypzin. See ZINEB.
Lannate. See METHOMYL.
Lanstan. See CHLORONITROPROPANE.
LEAD ARSENATE ... 50
  Other names: Gypsine, Soprabel
  Action: Insecticide.
Lebaycid. See FENTHION.
Legumex Extra. See BENAZOLIN.
Le-Kun. See DIMETHOATE.
LENACIL ... 111
  Chemical name: 3-cyclohexyl-5,6-trimethyleneuracil
  Other name: Venzar
  Action: Herbicide.
LeyCornox. See BENAZOLIN.

Page

LFN 111
Action: Herbicide.
LIME SULFUR 140
Chemical name: aqueous solution of calcium polysulfides
Action: Fungicide, Insecticide.
Lindafor. See LINDANE.
Lindagam. See LINDANE.
LINDANE 51
Chemical name: gamma isomer of 1,2,3,4,5,6-hexachloro-cyclohexane; also known as gamma benzene hexachloride
Other names: Gamaphex, Gamma BHC, Gammalin, Gammex, Gammexane, Isotox, Lindafor, Lindagam, Lintox, Novigam, Silvanol
Action: Insecticide.
Line Rider. See 2,4,5-T.
Lintox. See LINDANE.
LINURON 111
Chemical name: 3-(3,4-dichlorophenyl)-1-methoxy-1-methylurea
Other names: Afalon, Lorox, Sarclex
Action: Herbicide.
Liquiphene. See PMA.
Lonacol. See ZINEB.
Lorox. See LINURON.
Mafu. See DICHLORVOS.
Malachite. See COPPER CARBONATE.
MALACHITE GREEN 140
Chemical name: tetramethyl diapara-amidotriphenyl carbinol
Action: Fungicide.
Malamar. See MALATHION.
Malaspray. See MALATHION.
MALATHION 53
Chemical name: O,O-dimethyl S-(1,2-dicarbethoxyethyl) dithiophosphate
Other names: Carbofos, Carbophos, Cythion, Emmatos, Karbofos, Kop-Thion, Kypfos, Malamar, Malaspray, Malathon, Mercaptothion, Sithiol
Action: Insecticide.
Malathon. See MALATHION.
MALEAMIC ACID 140
Action: Fungicide.
MALEIC HYDRAZIDE 112
Chemical name: 1,2,3,6-tetrahydro-3,6-dioxopyridazine
Other names: KMH, Retard, Royal MH-30, Slo-Gro, Sprout-Stop, Sucker Stuff, Vandalhyde
Action: Herbicide.
Malix. See ENDOSULFAN.
Malis. See ENDOSULFAN.
Marlate. See METHOXYCHLOR.
Marmer. See DIURON.
Marvex. See DICHLORVOS.
Matacil. See AMINOCARB.
MBC. See SODIUM CHLORATE.

Page

MCA-600. See MOBAM.
MCP. See MCPA.
MCPA 112
Chemical name: 4-chloro-2-methylphenoxyacetic acid
Other names: Agroxone, Chiptox, Hormotuho, Kilsem, MCP, Mephanac, Metaxon, Methoxone, Rhomene, Rhonox
Action: Herbicide.
MCPB 112
Chemical name: 4-(4-chloro-2-methylphenoxy) butyric acid
Other names: 2M-4Kh-M, Thistrol, Tropotox
Action: Herbicide.
MECARBAM 55
Chemical name: *S*-*N*-ethoxycarbonyl-*N*-methylcarbamoyl-methyl *O*,*O*-diethylphosphorodithioate
Other names: Afos, Murfotox, Murotox, Pestan
Action: Insecticide.
Mediben. See DICAMBA.
Melprex. See DODINE.
MENAZON 55
Chemical name: *S*-(4,6-diamino-s-triazin-2-ylmethyl)*O*,*O*-dimethylphosphorodithioate
Other names: Azidithion, Saphi-Col, Saphizon, Saphos, Sayfos, Sayphos
Action: Insecticide.
Mendrin. See ENDRIN.
MEP. See FENITROTHION.
Mephanac. See MCPA.
Mercaptophos. See DEMETON, FENTHION.
Mercaptothion. See MALATHION.
Mercuram. See THIRAM.
MERCURY 140
Action: Fungicide.
MERPHOS 114
Chemical name: tributyl phosphorotrithioite
Other names: Deleaf Defoliant, Easy Off-D, Folex
Action: Herbicide.
Mersolite. See PMA.
METACIDE 55
Chemical name: mixture of 20% parathion and 80% of the dimethyl homologue
Action: Insecticide.
Metasystemox. See OXYDEMETON-METHYL.
Meta-Systox. See DEMETON METHYL.
Meta-Systox-R. See OXYDEMETON-METHYL.
Metaxon. See MCPA.
METHIONINE 152
Chemical name: α-amino-γ-methylthiolbutyric acid
Action: Fungicide.
METHOMYL 56
Chemical name: *S*-methyl-*N*-((methylcarbamoyl)oxy)thioacetimidate
Other name: Lannate
Action: Insecticide.
Methoxone. See MCPA.

Page
METHOXYCHLOR 58
Chemical name: 1,1,1-trichloro-2,2-bis(p-methoxyphenyl)ethane
Other names: Dianisyltrichloroethane, Dimethoxy-DT, DMDT, Marlate, Methoxy DDT
Action: Insecticide.
Methoxy DDT. See METHOXYCHLOR.
Methyl demeton. See DEMETON METHYL.
Methyl guthion. See AZINPHOS-METHYL.
Methylmercuric cyanoguanidine. See CYANO (METHYLMERCURI)GUANIDINE.
Methyl systox. See DEMETON METHYL.
METIRAM 153
Chemical name: common name for a group of fungicides based on polyethylene thiuram sulfide
Other names: [illegible], Polyram-Combi, Thioneb, Trioneb
Action: Fungicide.
MEVINPHOS 57
Chemical name: 2-methoxycarbonyl-1-methylvinyl dimethyl phosphate
Other names: Phosdrin, Phosfene
Action: Insecticide.
Mezineb. See PROPINEB.
Micofume. See DMTT.
Midox. See CHLORBENSIDE.
Milban. See ZIRAM.
MILBEX 58
Chemical name: mixture of 4-chlorophenyl 2,4,5-trichlorophenyl azosulfide and 1,1-bis(4-chlorophenyl)ethanol
Action: Insecticide.
Mildex. See DINOCAP.
Milogard. See PROPAZINE.
Mintacol. See PARAOXON.
MIREX 58
Chemical name: dodecachlorooctahydro-1,3,4-metheno-2*H*-cyclobuta(cd)pentalene
Other name: Dechlorane
Action: Insecticide.
Mitis green. See PARIS GREEN.
Mitox. See CHLORBENSIDE.
2M-4Kh-M. See MCPB.
MOBAM 59
Chemical name: 4-benzothienyl *N*-methylcarbamate
Other name: MCA-600
Action: Insecticide.
MOLINATE 118
Chemical name: *S*-ethyl *N*-hexahydro-1*H*-azepinethio-carbamate
Other names: Hydram, Ordram
Action: Herbicide.
Monobor-chlorate. See SODIUM CHLORATE.
MONOCROTOPHOS 59
Chemical name: 3-hydroxy-*N*-methyl-*cis*-crotonamide dimethyl phosphate
Other names: Azodrin, Nuvacron
Action: Insecticide.

Page
MONOXONE 116
Chemical name: Monochloroacetic acid
Action: Herbicide.
MONURON 116
Chemical name: 3-(p-chlorophenyl)-1,1-dimethylurea
Other names: Chlorfenidim, Telvar
Action: Herbicide.
Morestan. See OXYTHIOQUINOX.
Morocide. See BINAPACRYL.
MORPHOTHION 59
Chemical name: O,O-dimethyl S-(morpholinocarbonylmethyl)phosphorodithioate
Other names: Ekatin M, Morphotox
Action: Insecticide.
Morphotox. See MORPHOTHION.
Morsodren. See CYANO(METHYLMERCURI)GUANIDINE.
Murfotox. See MECARBAM.
Murotox. See MECARBAM.
Muscatox. See COUMAPHOS.
Mylone. See DMTT.
MYSTOX 155
Action: Fungicide.
Nabac. See HEXACHLOROPHENE.
NABAM 153
Chemical name: disodium ethylene-1,2-bisdithiocarbamate
Other names: Chem Bam, Dithane A-40, Dithane D-14, DSE, Parzate, Spring-Bak
Action: Fungicide.
NALCO 153
Action: Fungicide.
NALED 59
Chemical name: 1,2-dibromo-2,2-dichloroethyl dimethylphosphate
Other name: Dibrom
Action: Insecticide.
Nankor. See RONNEL.
NAPHTHA 118
Chemical name: light oil fraction from petroleum distillation
Action: Herbicide.
NAPHTHALENESULFONIC ACID 153
Action: Fungicide.
Naramycin A. See CYCLOHEXIMIDE.
Navadel. See DIOXATHION.
Neburea. See NEBURON.
NEBURON 116
Chemical name: 1-n-butyl-3-(3,4-dichlorophenyl)-1-methylurea
Other names: Kloben, Neburea
Action: Herbicide.
Neguvon. See TRICHLORFON.
Nemafume. See DIBROMOCHLOROPROPANE.
Nemagon. See DIBROMOCHLOROPROPANE.
Neobor. See BORAX.
Neocid. See DDT.
Nexion. See BROMOPHOS.
Niagaramite. See ARAMITE.
Niagaratran. See OVEX.

Page
Niagrathal. See ENDOTHALL.
Nialate. See ETHION.
NICOTINE 60
Chemical name: 1,3-(1-methyl-2-pyrrolidyl)pyridine
Action: Insecticide.
Nicouline. See ROTENONE.
Niran. See PARATHION.
Nitrador. See DNOC.
Nitropone C. See DNBP.
Nitrostigmine. See PARATHION.
N-METHYL CARBAMATE 60
Action: Insecticide.
Nogos. See DICHLORVOS.
Nomersan. See THIRAM.
No-Pest. See DICHLORVOS.
Novigam. See LINDANE.
Nuvacron. See MONOCROTOPHOS.
Nuvan. See DICHLORVOS.
Nuvanol. See FENITROTHION.
Octachlor. See CHLORDANE.
Octachlorocamphene. See TOXAPHENE.
Octa-Klor. See CHLORDANE.
Octalene. See ALDRIN.
Octalox. See DIELDRIN.
Octamethylpyrophosphoramide. See SCHRADAN.
Oko. See DICHLORVOS.
OMPA. See SCHRADAN.
Ordram. See MOLINATE.
Orthocide 406. See CAPTAN.
Ortho-Klor. See CHLORDANE.
Orthophos. See PARATHION.
Ortho phosphate defoliant. See DEF.
ORTHOZID 153
Action: Fungicide.
Ovatran. See OVEX.
OVEX 60
Chemical name: p-chlorophenyl p-chlorobenzenesulfonate
Other names: Chlorfenson, Chlorofenizon, CPCBS, Difenson, Ephirsulphonate, Estonmite, Niagaratran, Ovatran, Ovochlor, Ovotran, Sappiran, Trichlorfenson
Action: Insecticide.
Ovochlor. See OVEX.
Ovotran. See OVEX.
Oxine-copper. See COPPER 8-QUINOLINOLATE.
OXYDEMETON-METHYL 60
Chemical name: O,O-dimethyl S-[2-(ethylsulfinyl)ethyl]phosphorothioate
Other names: Metasystemox, Meta-Systox-R
Action: Insecticide.
OXYTETRACYCLINE 153
Chemical name: 4-dimethylamino-1,4,4a,5,5a,6,11,12a-octahydro-3,5,6,10,12,12a-hexahydroxy-6-methyl-1,11-dioxo-2-naphthacenecarboxamide
Other names: Biostat PA, Terramycin
Action: Fungicide.

Page
OXYTHIOQUINOX 61
Chemical name: 6-methyl-2,3-quinoxalinedithiol cyclic S,S-dithiocarbonate
Other names: Chinomethionate, Forstan, Morestan, Quinomethionate
Action: Insecticide, Fungicide.
Pallethrine. See ALLETHRIN.
Pamosol 2 Forte. See ZINEB.
Panodrin A-13. See CYANO(METHYLMERCURI)GUANIDINE.
Panogen. See CYANO(METHYLMERCURI)GUANIDINE.
Panoram D-31. See DIELDRIN.
Panthion. See PARATHION.
Paracide. See PARA-DICHLOROBENZENE.
PARA-DICHLOROBENZENE 153
Chemical name: 1,4-dichlorobenzene
Other names: Paracide, Paradow, PDB, Santochlor
Action: Fungicide.
Paradow. See PARA-DICHLOROBENZENE.
Paramar. See PARATHION.
PARAOXON 61
Chemical name: O,O-diethyl O-p-nitrophenyl phosphate
Other name: Mintacol
Action: Insecticide.
Paraphos. See PARATHION.
PARAQUAT 116
Chemical name: 1,1'-dimethyl-4,4'-bipyridynium ion
Other names: Gramoxone, Weedol
Action: Herbicide.
Parathene. See PARATHION.
PARATHION 61
Chemical name: O,O-diethyl O-p-nitrophenyl phosphorothioate
Other names: AAT, Alkron, Alleron, Aphamite, Corothion, DNTP, Ethyl Parathion, Etilon, Folidol, Niran, Nitrostigmine, Orthophos, Panthion, Paramar, Paraphos, Parathene, Parawet, Phoskil, Rhodiatox, SNP, Soprathion, Stathion, Thiophos
Action: Insecticide.
Parawet. See PARATHION.
PARIS GREEN 64
Chemical name: copper acetoarsenite
Other names: Emerald green, French green, Mitis green, Schweinfurt green
Action: Insecticide.
Parzate. See NABAM.
Parzate C. See ZINEB.
Parzate Zineb. See ZINEB.
PCA. See PYRAZON.
PCB. See AROCHLORS.
PCNB 153
Chemical name: pentachloronitrobenzene
Other names: Avicol, Botrilex, Brassicol, Folosan, PKhNB, Quintozene, Terrachlor, Terraclor, Tilcarex, Tri-PCNB, Tritisan
Action: Fungicide.

Page
PCP 117
Chemical name: pentachlorophenol
Other names: Penchlorol, Penta, Santobrite, Santophen 20, Sinituho, Weedone
Action: Herbicide, Fungicide, Insecticide.
PDB. See PARA-DICHLOROBENZENE.
PDU. See FENURON.
Penchlorol. See PCP.
Penite. See SODIUM ARSENITE.
Penta. See PCP.
Pentachlorin. See DDT.
Perfekthion. See DIMETHOATE.
PERTHANE 64
Chemical name: 1,1-dichloro-2,2bis(4-ethylphenyl)ethane
Action: Insecticide.
Pestan. See MECARBAM.
Pestox III. See SCHRADAN.
Phaltan. See FOLPET.
Phenacide. See TOXAPHENE.
Phenatox. See TOXAPHENE.
PHENETIDINE 153
Chemical name: ortho, meta, or para-aminophenate
Action: Fungicide.
Phenmad. See PMA.
Phenol. See CARBOLIC ACID.
PHENOXYTOL 154
Action: Fungicide.
Phix. See PMA.
PHORATE 64
Chemical name: *O,O*-diethyl *S*-(ethylthio)-methyl phosphorodithioate
Other names: Thimet, Timet
Action: Insecticide.
PHOSALONE 65
Chemical name: *O,O*-diethyl *S*-(6-chlorobenzoxazolone-3-yl-methyl)phosphorodithioate
Other name: Zolone
Action: Insecticide.
Phosdrin. See MEVINPHOS.
Phosfene. See MEVINPHOS.
Phoskil. See PARATHION.
PHOSPHAMIDON 65
Chemical name: 2-chloro-*N,N*-diethyl-3-hydroxycrotonamide dimethylphosphate
Other names: Dimecron, Dimicron
Action: Insecticide.
Phosvit. See DICHLORVOS.
Phygon. See DICHLONE.
PICLORAM 118
Chemical name: 4-amino-3,5,6-trichloropicolinic acid
Other names: Borolin, Tordon
Action: Herbicide.
PIPERONYL BUTOXIDE 66
Chemical name: α[2-(2-butoxyethoxy)ethoxy]-4,5-(methylenedioxy)-2-propyltoluene; also butyl carbityl 6-propylpiperonyl ether
Other name: Butacide
Action: Insecticide.

Page
PKhNB. See PCNB.
PMA 154
Chemical name: phenylmercury acetate
Other names: Gallotox, Hong Nien, Liquiphene, Mersolite, Phenmad, Phix, PMAS, Shimmerex
Action: Fungicide, Herbicide.
PMAS. See PMA.
Polybor chlorate. See SODIUM CHLORATE.
Polychlorinated biphenyls. See AROCHLORS.
Polychlorocamphene. See TOXAPHENE.
Polyram-Combi. See METIRAM.
Polyram-Ultra. See THIRAM.
Polyram Z. See ZINEB.
Pomarsol Z Forte. See ZIRAM.
Pomasol Forte. See THIRAM.
POTASSIUM PERMANGANATE 154
Action: Fungicide.
Pramitol. See PROMETONE.
PRB 154
Action: Fungicide.
Prefix. See CHLORTHIAMID.
Premerge. See DNBP.
Preventol. See DICHLOROPHEN.
Preservit. See DMTT.
Primatol A. See ATRAZINE.
Primatol P. See PROPAZINE.
Princep. See SIMAZINE.
Prolan-Bulan Mixture. See DILAN.
Prometon. See PROMETONE.
PROMETONE 119
Chemical name: 2-methoxy-4,6-bis(isopropylamino)-*s*-triazine
Other names: Gesafram, Pramitol, Prometon
Action: Herbicide.
PROMETRYNE 120
Chemical name: 2,4-bis(isopropylamino)-6-methylmercapto-*s*-triazine
Other names: Caparol, Gesagard
Action: Herbicide.
PROPAZINE 120
Chemical name: 2-chloro-4,6-bis(isopropylamino)-*s*-triazine
Other names: Gesamil, Milogard, Primatol P
Action: Herbicide.
PROPHAM 120
Chemical name: isopropyl carbanilate
Other names: Chem-Hoe, IFC, IPC, Triherbicide, Tuberite
Action: Herbicide.
PROPINEB 154
Chemical name: zinc propylenebisdithiocarbamate
Other names: Antracol, Mezineb
Action: Fungicide.
PROPOXUR 66
Chemical name: *o*-isopropoxyphenyl methylcarbamate
Other names: Arprocarb, Baygon, Blattanex, Suncide, Unden
Action: Insecticide.
Pynamin. See ALLETHRIN.

Page

Pyramin. See **PYRAZON**.

PYRAZON 181

Chemical name: 5-amino-4-chloro-2-phenyl-3-pyridazinone

Other names: PCA, Pyramin

Action: Herbicide.

PYRETHRINS 66

Chemical name: insecticidal compounds in pyrethrum flowers, *Chrysanthemum cinerariaefolium*

Other names: Cinerins, Jasmolins

Action: Insecticide.

Pyrimithate. See **DICTHYL**.

Quelatox. See **FENTHION**.

Queletox. See **FENTHION**.

Quilan. See **BENEFIN**.

QUININE 184

Action: Fungicide.

Quinomethionate. See **OXYTHIOQUINOX**.

Quintozene. See **PCNB**.

Rabon. See **GARDONA**.

Radapon. See **DALAPON**.

Randox. See **CDAA**.

Rasikal. See **SODIUM CHLORATE**.

Reddon. See **2,4,5-T**.

Reglone. See **DIQUAT**.

REGULOX 181

Action: Herbicide.

Resitox. See **COUMAPHOS**.

Retard. See **MALEIC HYDRAZIDE**.

Rhodiatox. See **PARATHION**.

Rhomene. See **MCPA**.

Rhonox. See **MCPA**.

Rhothane. See **TDE**.

Rogor. See **DIMETHOATE**.

RONNEL 67

Chemical name: O,O-dimethyl O-2,4,5-trichlorophenyl phosphorothioate

Other names: Etrolene, Fenchlorfos, Korlan, Nankor, Trolene, Viozene

Action: Insecticide.

Rospin. See **CHLOROPROPYLATE**.

ROTENONE 67

Chemical name: extracts from *Derris* or *Lonchocarpus* plants

Other names: Derrin, Nicouline, Tubatoxin

Action: Insecticide.

Roxion. See **DIMETHOATE**.

Royal MH-30. See **MALEIC HYDRAZIDE**.

Ruelene. See **CRUFOMATE**.

Rukseam. See **DDT**.

Ruphos. See **DIOXATHION**.

SAFROLE 184

Chemical name: 4-allyl-1,2-methylene dioxybenzene

Action: Fungicide.

SALICYLIC ACID 184

Action: Fungicide.

Santobrite. See **PCP**.

Santochlor. See **PARADICHLOROBENZENE**.

Santophen 20. See **PCP**.

Page

Sapecron. See **CHLORFENVINPHOS**.

Saphi-Col. See **MENAZON**.

Saphizon. See **MENAZON**.

Saphos. See **MENAZON**.

Sappiran. See **OVEX**.

Sarclex. See **LINURON**.

Sayfos. See **MENAZON**.

Sayphos. See **MENAZON**.

SCHRADAN 68

Chemical name: bis-N,N,N',N'-tetramethylphosphorodiamidic anhydride

Other names: Octamethylpyrophosphoramide, OMPA, Pestox III, Sytam

Action: Insecticide.

Schweinfurt green. See **PARIS GREEN**.

Seedrin. See **ALDRIN**.

Semesan. See **HYDROXYMERCURICHLOROPHENOLS**.

SES. See **SESONE**.

SESONE 181

Chemical name: sodium 2,4-dichlorophenoxyethyl sulfate

Other names: 2,4-DES-Na, Disul-Na, SES

Action: Herbicide.

Sevin. See **CARBARYL**.

Shed-A-Leaf. See **SODIUM CHLORATE**.

Shimmerex. See **PMA**.

Silvanol. See **LINDANE**.

SILVEX 181

Chemical name: 2-(2,4,5-trichlorophenoxy)propionic acid

Other names: Esteron, Fenoprop, Garlon, Kuron, Kurosal, 2,4,5-TP

Action: Herbicide.

Silvisar 510. See **CACODYLIC ACID**.

SIMAZINE 183

Chemical name: 2-chloro-4,6-bis (ethylamino)-s-triazine

Other names: Gesatop, Princep

Action: Herbicide.

Sinituho. See **PCP**.

Sinox. See **DNOC**.

Sinox General. See **DNBP**.

Slo-Gro. See **MALEIC HYDRAZIDE**.

SMDC 184

Chemical name: sodium N-methyldithiocarbamate

Other names: Trimaton, Vapam, VPM

Action: Fungicide, Herbicide.

SNP. See **PARATHION**.

SODIUM ARSENITE 184

Chemical name: sodium meta-arsenite

Other names: Atlas 'A', Chem Pels C, Penite, Sodium meta-arsenite

Action: Herbicide, Insecticide.

SODIUM CHLORATE 185

Other names: Atlacide, Atratol, Chlorax, De-Fol-Ate, Drop-Leaf, Fall, KMBC, Monobor-chlorate, Polybor Chlorate, Rasikal, Shed-A-Leaf, Tumbleaf

Action: Herbicide.

Page
Sodium meta-arsenite. See SODIUM ARSENITE.
SODIUM NITRITE 155
  Action: Fungicide.
SODIUM PENTACHLOROPHENATE 155
  Other names: Dowicide G, Weedbeads
  Action: Herbicide.
Sodium TCA. See TCA.
Solvirex. See DISULFOTON.
Soprabel. See LEAD ARSENATE.
Sopranthion. See PARATHION.
Spectracide. See DIAZINON.
Spergon. See CHLORANIL.
Spotrete. See THIRAM.
Spring-Bak. See NABAM.
Sprout Nip. See CHLORPROPHAM.
Sprout-Stop. See MALEIC HYDRAZIDE.
Stathion. See PARATHION.
Strobane. See TERPENE POLYCHLORINATES.
Strobane-T. See TOXAPHENE.
Sucker Stuff. See MALEIC HYDRAZIDE.
Sulfallate. See CDEC.
SULFUR 155
  Other name: Brimstone
  Action: Fungicide, Insecticide.
Sumithion. See FENITROTHION.
Sumitomo. See FENITROTHION.
Suncide. See PROPOXUR.
Supona. See CHLORFENVINPHOS.
Synklor. See CHLORDANE.
Systox. See DEMETON.
Sytam. See SCHRADAN.
2,4,5-T 156
  Chemical name: 2,4,5-trichlorophenoxyacetic acid
  Other names: Ded-Weed Brush Killer, Esteron 245 Concentrate, Fence Rider, Inverton 245, Line Rider, Reddon
  Action: Herbicide.
TAR DISTILLATE 157
  Chemical name: product of fractional distillation of tars
  Action: Herbicide.
2,3,6-TBA 156
  Chemical name: 2,3,6-trichlorobenzoic acid
  Other names: Benzac, Fen-All, Tribac, Trichlorobenzoic acid, Trysben 200, Zobar
  Action: Herbicide.
TBTO. See TRIBUTYL TIN OXIDE.
TCA 156
  Chemical name: trichloroacetic acid
  Other name: Sodium TCA
  Action: Herbicide.
TDE 66
  Chemical name: 2,2-bis(p-chlorophenyl)-1,1-dichloroethane
  Other names: DDD, Rhothane
  Action: Insecticide.
Tedion. See TETRADIFON.
Telvar. See MONURON.
Tenoran. See CHLOROXURON.
TEP. See TEPP.

Page
TEPP 69
  Chemical name: tetraethyl pyrophosphate and other ethyl phosphates
  Other names: Bladan, HETP, Kilmite 40, TEP, Tetron, Vapotone
  Action: Insecticide.
TERPENE POLYCHLORINATES 70
  Chemical name: polychlorinates of camphene, pinene, and related terpenes
  Other name: Strobane
  Action: Insecticide.
Terrachlor. See PCNB.
Terraclor. See PCNB.
Terracur. See FENSULFOTHION.
Terramycin. See OXYTETRACYCLINE.
Tersan. See HYDROXYMERCURICHLOROPHENOLS, THIRAM.
TETRACYCLINE HYDROCHLORIDE 156
  Action: Fungicide.
TETRADIFON 70
  Chemical name: p-chlorophenyl 2,4,5-trichlorophenyl sulfone
  Other names: Duphar, Tedion
  Action: Insecticide.
Tetron. See TEPP.
THANITE 70
  Chemical name: isobornyl thiocyanoacetate
  Action: Insecticide.
Thifor. See ENDOSULFAN.
Thimer. See THIRAM.
Thimet. See PHORATE.
Thimul. See ENDOSULFAN.
Thiodan. See ENDOSULFAN.
Thiodemeton. See DISULFOTON.
Thioneb. See METIRAM.
Thiophal. See FOLPET.
Thiophos. See PARATHION.
Thiosan. See THIRAM.
Thiotex. See THIRAM.
THIOUREA 158
  Other names: Thiocarbamide, Sulfourea
  Action: Fungicide.
THIRAM 158
  Chemical name: bis(dimethylthiocarbamoyl)disulfide or tetramethylthiuram disulfide
  Other names: Arasan, Fernide 850, Hexathir, Mercuram, Nomersam, Polyram-Ultra, Pomasol Forte, Spotrete, Tersan, Thimer, Tidesan, Thiotex, Thiramad, Thirasan, Thylate, Tirampa, TMTD, TMTDS, Trametan, Tripomol, Tuads
  Action: Fungicide.
Thiramad. See THIRAM.
Thirasan. See THIRAM.
Thistrol. See MCPB.
Thylate. See THIRAM.
THYMOL 157
  Chemical name: 5-methyl-2-isopropylphenol
  Action: Fungicide.
Tiezene. See ZINEB.
Tiguvon. See FENTHION.

Page
Tilcarex. See PCNB.
Timet. See PHORATE.
Tirampa. See THIRAM.
TMTD. See THIRAM.
TMTDS. See THIRAM.
Tomatotone. See 4-CPA.
Topiclor 20. See CHLORDANE.
Tordon. See PICLORAM.
Toxakil. See TOXAPHENE.
TOXAPHENE 70
  Chemical name: mixture of various chlorinated camphenes
  Other names: Alltox, Chlorinated camphene, Octachlorocamphene, Phenacide, Phenatox, Polychlorocamphene, Strobane-T, Toxakil
  Action: Insecticide.
2,4,5-TP. See SILVEX.
Trametan. See THIRAM.
Treflan. See TRIFLURALIN.
Trifocide. See DNOC.
TRIAMIPHOS 157
  Chemical name: p-(5-amino-3-phenyl-1H-1,2,4-triazol-1-yl)-N,N,N',N'-tetramethyl phosphonic diamide
  Other name: Wepsyn
  Action: Fungicide, Insecticide.
Triasyn. See DYRENE.
Tribac. See 2,3,6-TBA.
TRIBUTYL TIN OXIDE 157
  Chemical name: bis(tri-n-butyltin) oxide
  Other names: Butinox, TBTO
  Action: Fungicide.
Tricarbamix. See FERBAM, ZINEB.
Tricarbamix Z. See ZIRAM.
Trichlorfenson. See OVEX.
TRICHLORFON 74
  Chemical name: dimethyl (2,2,2-trichloro-1-hydroxyethyl) phosphonate
  Other names: Anthon, Chlorofos, Dipterex, Dylox, Neguvon, Trichlorphon, Tugon
  Action: Insecticide.
Trichlorobenzoic acid. See 2,3,6-TBA.
Trichloromethylthiophthalimide. See FOLPET.
Trichlorophon. See TRICHLORFON.
Tridodine. See DODINE.
Tri-Endothal. See ENDOTHALL.
Tri-Fen. See FENAC.
Tri-Fene. See FENAC.
TRIFLURALIN 128
  Chemical name: α,α,α-trifluoro-2,6-dinitro-N,N-dipropyl-p-toluidine
  Other names: Elancolan, Treflan
  Action: Herbicide.
Trifungol. See FERBAM.
Triherbide. See PROPHAM.
Trimaton. See SMDC.
Trimetion. See DIMETHOATE.
Triofterol. See ZINEB.
Triscabol. See METIRAM.
TRIOXONE 129
  Action: Herbicide.

Page
Tri-PCNB. See PCNB.
Tripomol. See THIRAM.
Triscabol. See ZIRAM.
Trithion. See CARBOPHENOTHION.
Tritisan. See PCNB.
Tri-VC 13. See DICHLOFENTHION.
Trolene. See RONNEL.
Tronabor. See BORAX.
Tropotox. See MCPB.
Trysben 200. See 2,3,6-TBA.
Tuads. See THIRAM.
Tubatoxin. See ROTENONE.
Tuberite. See PROPHAM.
Tugon. See TRICHLORFON.
Tumbleaf. See SODIUM CHLORATE.
Unden. See PROPOXUR.
Unipon. See DALAPON.
UREABOR 129
  Chemical name: complex of sodium metaborate/chlorate and bromacil
  Other name: BMM
  Action: Herbicide.
Vamidoate. See VAMIDOTHION.
VAMIDOTHION 74
  Chemical name: O,O-dimethyl-S-(2((1-methylcarbamoyl-ethyl)thio)ethyl)phosphorothioate
  Other names: Kilval, Vamidoate
  Action: Insecticide.
Vandalhyde. See MALEIC HYDRAZIDE.
Vapam. See SMDC.
Vapona. See DICHLORVOS.
Vapotone. See TEPP.
VC-13 Nemacide. See DICHLOFENTHION.
Vegadex. See CDEC.
Velsicol 1068. See CHLORDANE.
Venzar. See LENACIL.
Vernam. See VERNOLATE.
VERNOLATE 129
  Chemical name: S-propyl-N,N-dipropylthiocarbamate
  Other name: Vernam
  Action: Herbicide.
Viozene. See RONNEL.
Viricuivre. See COPPER OXYCHLORIDE.
VPM. See SMDC.
Weed-Ag-Bar. See 2,4-D.
Weedar 64. See 2,4-D.
Weedazol. See AMITROLE.
Weedbeads. See SODIUM PENTACHLOROPHENATE.
Weed-B-Gon. See 2,4-D.
Weedol. See PARAQUAT.
Weedone. See PCP, 2,4-D.
Y-3. See CHLORPROPHAM.
Zebtox. See ZINEB.
ZECTRAN 75
  Chemical name: 4-dimethylamino-3,5-xylyl N-methylcarbamate
  Action: Insecticide.
Zerdane. See DDT.
Zerlate. See ZIRAM.

Page

ZINC CHLORIDE 76
  Other name: Butter of zinc
  Action: Insecticide, Fungicide.
ZINC HYDROXYQUINONE 187
  Action: Fungicide.
ZINEB 187
  Chemical name: zinc ethylene-1,2-bisdithiocarbamate
  Other names: Aspor, Chem Zineb, Dithane Z-78, Hexathane, Kypzin, Lonacol, Pamosol 2 Forte, Parzate C, Parzate zineb, Polyram Z, Tiezene, Tricarbamix, Triofersol, Zebtox, Zinosan
  Action: Fungicide.

Page

Zinosan. See ZINEB.
ZIRAM 188
  Chemical name: zinc dimethyldithiocarbamate
  Other names: Cuman, Fuklasin, Hexazir, Milbam, Pomarsol Z Forte, Tricarbamix Z, Triscabol, Zerlate, Zirberk, Ziride, Zitox
  Action: Fungicide.
Zirberk. See ZIRAM.
Ziride. See ZIRAM.
Zithiol. See MALATHION.
Zitox. See ZIRAM.
Zobar. See 2,3,6-TBA.
Zolone. See PHOSALONE.
Zytron. See DMPA.

Subject Index

Abortion: in reindeer, 66
—— in mosquito fish, 170; caused by aldrin, 4; DDT, 21; lindane, 51; methoxychlor, 56; TDE, 62; toxaphene, 72
Absorbing capacity of plants, 69-70
Acari. *See* Mite
Acetylcholinesterase, 74
Actinomycete, 60
Adelie penguin, residues in, 166, 167
Adrenal weight, 16
Aerobic organism, growth of, 60
Age affecting mercury content, 151
Air, residues in, 172-173
Aircraft spraying, 151
Alewife, biological concentration in, 27
Alfalfa: affected, 30, 110; residues in, 77; translocation into, 5, 30
Algae: biological concentration in, 6, 36, 37, 44, 72; composition of community altered, 86; converting malathion, 55; growth, 11, 66, 170; mortality, 11, 115, 120; populations decreased, 147; productivity, 60; unaffected, 27, 101, 110. *See also* Blue-green algae; Diatom; Periphyton
Alkalinity of water, effect on LC_{50}, 141
American redstart population reduced, 10
American smelt, residues in, 160
American sparrow hawk: eggshell thinning in, 17, 19, 173; sensitivity to DDT, 177
Amino acids in plants, 87, 94, 112
Ammonification, 7, 92, 99
Amphipod: biological concentration in, 27; no mortality in, 115; population numbers, 86, 125; residues in, 27
—— LC_{50} to: abate, 2; aldrin, 3; allethrin, 6; aminocarb, 6; aramite, 7; azide, 95; azinphos-methyl, 8; captafol, 155; carbaryl, 11; carbophenothion, 12; chlordane, 13; chloronitropropane, 140; Ciodrin, 15; coumaphos, 15; 2,4-D, 20; DDT, 24; DEF, 109; Dexon, 148; diazinon, 30; dicamba, 102; dichlobenil, 103; dichlone, 144; dichlorvos, 32; dicrotophos, 33; dieldrin, 36; Dilan, 37; dimethoate, 38; dioxathion, 39; disulfoton, 39; diuron, 107; dursban, 41; endosulfan, 42; endothall, 108; endrin, 44; EPN, 44; EPTC, 45; ethion, 45; fenac, 110; fenthion, 47; heptachlor, 49; lindane, 52; malathion, 54; methoxychlor, 57; mevinphos, 58; molinate, 114; naled, 60; naphtha, 115; oxydemetonmethyl, 60; paraquat, 117; parathion, 63; phorate, 64, 65; phosphamidon, 66; picloram, 118; propham, 120; propoxur, 66; pyrethrins, 67; rotenone, 68; simazine, 120; TDE, 69; TEPP, 69; tetradifon, 70; toxaphene, 73; trichlorfon, 74; trifluralin, 120; vernolate, 120; zectran, 75
Anaerobic organism, growth of, 60
Anchovy, residues in, 166
Anisoptera. *See* Dragonfly
Ant population eliminated, 107
Antelope, residues in, 163, 164
Aphid: longevity unaffected, 97; populations increased, 24, 25, 26, 28, 97, 170
Apple tree, translocation in, 151
Aquatic worm. *See* Oligochaete
Ascomycete growth reduced, 148
Ash susceptibility, 115
Ashy petrel, residue in, 167-168
Asiatic clam, biological concentration in, 60
Aspen, aerial portions affected, 80
Ataxia, 16
Atlantic croaker: biological concentration in, 27; mortality, 21
Atlantic gannet, residues in, 167
Atlantic salmon: change in diet, 25; mortality, 20-22; unaffected, 66
Atmosphere, residues in, 172-173
Attraction to mice, 20
Auk, residues in, 163
Azuki-bean weevil egg production reduced, 48

Bacteria: ammonifying, 6, 92, 99; growth, 93, 99, 112; nitrifying. *See* Nitrification; populations, 110, 124, 156-157; respiration inhibited, 127; toxicity to, 6, 13, 60. *See also* Actinomycete
Bald eagle: eggshell thinning, 17; eggshell weight, 17-18; LC_{50}, 17; poisoned, 17; population decline, 17; reproduction suppressed, 17; residues in, 17, 166, 167
Bark beetle: hatchability, 60; mortality, 60
Barley, affected, 30, 110
Barn owl, residues in, 166, 167
Basidiomycete growth reduced, 148
Bass: LC_{50}, 119; mortality, 105, 141. *See also* Striped bass
Bay mussel, EC_{50}, 10

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Bean plant: absorbing capacity of, 98-99; aluminum content of, 18; amino acid content of, 97, 99, 112; copper content of, 94; effect on yield of, 90, 91, 147, 155; growth affected, 90-97, 12, 49, 170; iron content of, 18, 94; nitrogen content of, 98, 98-99, 180; protein content of 98-99, 180; toxicity to, 90; wilt disease in, 105; zinc content of, 94, 180
Bear, residues in, 168, 164. *See also* Black bear
Beaver, residues in, 180
Beet yield reduced, 91
Beetle: biomass, 5, 99; populations, 5, 26, 56, 120-124, 131; residues in, 166, 170. *See also* Azuki bean weevil; Bark beetle; Coccinellid; Ground beetle; Hydrophilid; Water beetle; Weevil; Wireworm
Behavioral changes, 176-179; in bird, 46; fiddler crab, 99; [illegible] fish, 117; guppy, 49; pheasant, 54; salmon, 21, 34, 186; sheep, 99, 175; trout, 21, 74, 175-179
Bengalese finch: eggshell, 17; ovulation time, 17, 170
Bentgrass, nematodes in, 101, 119
Bermuda petrel, residues in, 168
Biological concentration, 169; in cranberry bog, 90; food chain, 151, 169; forest, 99; pond and lake, 27-28, 99, 169; annelids, 26, 36-37, 151; arthropods, 27, 28, 30-31, 73, 151; birds, 27-28, 99, 73; plankton, 28, 48, 73
—— in fish, 90, 93, 99; alewife, 27; black bullhead, 26; bluegill, 33, 58, 117, 126, 128; chinook salmon, 151; chub, 27; croaker, 27; fathead minnow, 33, 44; goldfish, 73; pike, 151; sunfish, 99, 100, 105; trout, 28, 36, 57, 73, 105; whitefish, 27
—— in mollusks, 26, 30, 99, 48, 151; oyster, 44, 53, 58, 87; eastern oyster, 18, 27, 37, 41, 99
—— in plants, 37, 90, 73, 151; algae, 5, 26, 27, 44, 73
Biomass, 5, 25, 99
Biotic community, 99
Bird: behavioral change, 46, 99; biological concentration in, 99; mortality, 99; repellence of, 130; residue loss in, 26; specificity of response, 46
Births reduced, 9, 16, 33, 42, 47, 56
Biting midge populations reduced, 56, 99, 135
Bitterbrush unharmed, 99
Bivalves, residues in, 172
Black bear populations unaffected, 45
Blackbird, repellence of, 129, 148, 158. *See also* Cowbird
Black bullhead: biological concentration in, 26; mortality, 29; resistance in, 4-5, 21, 35, 48, 73
—— LC_{50} to: aldrin, 3; azinphosmethyl, 8; carbaryl, 10; DDT, 20, 21; dieldrin, 35; endothall, 108; endrin, 43; fenthion, 46; lindane, 52; malathion, 56; parathion, 62; picloram, 118; toxaphene, 71, 72; zectran, 75
Black cherry tree, hydrocyanic acid content of, 96, 127
Black duck, residues in, 167
Black duck, residues in, 167
Black oak damaged, 99
Black tern reproduction stopped, 71
Blindness in fish, 146-147
Blue crab mortality, 36
Bluegill: biological concentration in, 33, 58, 117, 126, 128; blindness, 146-147; converting 2,4-DB to 2,4-D, 94; gill damaged, 103; growth, 46, 58, 96; kidney damaged, 124; LD_{50}, 124; liver damaged, 124; mortality, 94, 95, 141, 146, 147; pathologic lesions in, 96; spawning delayed, 94; resistance in, 4, 35, 48, 73, 109
—— EC_{50} to: azinphosmethyl, 8; carbaryl, 10; DDT, 20; diazinon, 28-29; dieldrin, 35; diuron, 106; endrin, 43; fenuron TCA, 111; heptachlor, 48; lindane, 51; malathion, 56; monuron, 114; toxaphene, 71
—— LC_{50} to: aldrin, 3; arsenite, 7; azide, 90; azinphosmethyl, 8; carbaryl, 10; carbophenothion, 12; chlordane, 13; chlorpropham, 91; copper oxychloride, 140; copper sulfate, 141; Cytrol Amitrole-T, 92; 2,4-D, 94, 95; dalapon, 100, 101; DDD, 99; DDT, 100; demeton, 99; dexon, 143; diazinon, 99; dicamba, 100; dichlobenil, 100; dichlorprop, 104; dichlorvos, 31; dieldrin, 35; dilan, 37; dimethoate, 38; dioxathion, 38; diquat, 103; disulfoton, 39; diuron, 106; Dyrene, 143; endothall, 108; endrin, 42, 43; EPN, 44; ethion, 45; fenac, 110; fenthion, 46; fentin hydroxide, 146; heptachlor, 48; lindane, 52; malathion, 56; MCPA, 112; mevinphos, 57; naled, 59; ovex, 60; paraquat, 116; parathion, 62; phorate, 64; picloram, 118; propham, 120; rotenone, 67; silvex, 121, 122; simazine, 125; sodium arsenite, 124; 2,4,5-T, 126-127; TDE, 99; terpene polychlorinates, 70; tetradifon, 70; toxaphene, 71, 72; trifluralin, 128; vernolate, 128; zectran, 75; affected by alkalinity of water, 141; affected by hardness of water, 141; affected by temperature. *See* Temperature affecting LC_{50} for bluegill
—— survival in concentration of: amitrole, 90; atrazine, 87; benzenethiol, 137; biuret, 137; carbolic acid, 138; cyclopentadiene, 142; 2,4-D, 94; dalapon, 100; dichlobenil, 102; diquat, 104; endothall, 108; fenac, 109; fenuron TCA, 111; hippuric acid, 149; maleamic acid, 149; maleic hydrazide, 113; methionine, 152; monuron, 114-115; naphthalenesulfonic acid, 155; paraquat, 116; phenetidine, 158; safrole, 154; silvex, 122; 2,4,5-T, 127
Blue-green algae, resistance in, 141
Blue grouse mortality, 85
Blue waxbill mortality, 69
Blue-winged teal: reproduction stopped, 12; residues in, 168
Bluntnose minnow: LC_{50}, 49, 108; mortality, 110; survival, 108; tolerance affected by hardness of water, 72
Bobwhite quail: growth, 110; LD_{50}, 4, 11-12, 46, 58, 70-71, 100; mortality, 46; poisoned, 150; repellence of, 130; reproduction depressed, 170
—— LD_{50} to: abate, 3; aldrin, 4; Azodrin, 7; atrazine, 87; azinphosmethyl, 8; cadmium succinate, 138; captan, 138; carbaryl, 9-10; chlordane, 13; 2,4-D, 96; DDD, 16; DDT, 18; demeton, 20; dicofol, 32; dieldrin, 35; disulfoton, 39; diuron, 105-106; endosulfan, 41; endrin, 42; EPN, 44-45; fensulfothion, 46; fenthion, 46; heptachlor, 47; lindane, 51; malathion, 55; methoxychlor, 56; naled, 59; parathion, 61; Paris green, 64; phorate, 64; phosphamidon, 65; TDE, 66; terpene polychlorinates, 70; toxaphene, 71; trichlorfon, 74
Boron content of bean plant, 97
Box elder susceptibility, 115
Box turtle unaffected, 23
Brain: pathologic effects on, 96; residues in, 17, 34, 66, 168
Brandt's cormorant, residues in, 167-168
Brill, residues in, 168
Broad-leaved plants, resistance in, 106

Brook trout: behavioral change in, 21; biological concentration in, 97; fin erosion, 147; growth, 21; LC_{50}, 8, 20, 58, 60, 111; mortality, 21, 22, 96, 147; unaffected, 22, 65, 100, 104
Brown bullhead: TL_{50}, 105; unaffected, 41, 46
Brown pelican: eggshell thinning, 20; reproduction, 20, 170; residues in, 20, 105
Brown shrimp: EC_{50}, 130; survival, 10, 72; unaffected, 106, 117, 118, 127
—— mortality or paralysis caused by: ametryne, 20; atrazine, 98; 2,4-D, 98; dalapon, 101; DDT, 24; lindane, 52; silvex, 132
Brown trout, residues in, 22
—— LC_{50} to: azinphos-methyl, 8; carbaryl, 10; DDT, 20; fenthion, 46; lindane, 52; malathion, 53; parathion, 60; picloram, 118; toxapene, 71; zectran, 76
—— survival in concentration of: benzenethiol, 137; biuron, 137; carbolic acid, 139; cyclopentadiene, 142; hippuric acid, 148; maleamic acid, 149; methionine, 152; naphthalenesulfonic acid, 150; phenetidine, 153; safrole, 154
Browse improved, 98
Buckwheat: nitrogen content of, 98; protein nitrogen content of, 98; sugar content of, 98
Buff-bellied chipmunk, populations unaffected, 16
Bug, populations unaffected, 125, 126, 133, 135
—— populations reduced by concentration of: binapacryl, 9; captan, 130; carbaryl, 11; dimethoate, 36; dinocap, 146; sulfur, 155; triamiphos 107
Bullfinch, residues in, 104
Bullfrog, mortality, 137, 140
—— LC_{50} to: abate, 3; DDT, 24; demeton, 30; diazinon, 30; dicrotophos, 33; phorate, 64; propoxur, 66; TEPP, 69; zectran, 76
Bullhead: LC_{50}, 108; residues in, 106. *See also* Black bullhead; Brown bullhead
Buzzard: mortality, 34; residues in, 150

Cabbage aphid population outbreak, 68
Caddice fly: biomass, 20; populations decreased, 20, 25, 26, 98, 115; populations increased, 7, 25; residues in, 106; survival, 107
Calcium in eggs, 18
California quail, LD_{50}, 65
Canada goose, LD_{50} to: carbaryl, 9; dicrotophos, 32; dieldrin, 34; dursban, 40; fenthion, 46; monocrotophos, 58; naled, 59; propoxur, 66; zectran, 75
Canada thistle: nitrate content of, 98; palatability, 98
Carabid. *See* Ground beetle
Carbonic anhydrase activity, 18
Caribou, residues in, 105, 106
Carp: mortality, 8, 22, 141; survival, 62
—— LC_{50} to: azinphos-methyl, 8; carabaryl, 10; DDT, 20; endothall, 100; endrin, 42; fenthion, 46; lindane, 52; malathion, 53; parathion, 62; toxaphene, 71; zectran, 75
Carrot yield, 20, 31, 147, 155
Cassin's auklet, residues in, 106
Cat, LD_{50}, 107
Caterpillar, natural enemies killed, 26
Catfish: population eliminated, 120; residues in, 105; survival, 141. *See also* Channel catfish
Cellulose decomposition, 22, 109, 112
Ceratopogonid. *See* Biting midge
Chaoborid. *See* Phantom midge
Channel catfish: EC_{50}, 10, 20, 25, 46, 58; unaffected, 110
—— LC_{50} to: azinphos-methyl, 8; captafol, 126; carbaryl, 10; carbolic acid, 139; cresol, 142; DDT, 20; Delnav, 140; dichlone, 144; fenthion, 46; ferbam, 146; formalin, 146; lindane, 52; malathion, 53; mercury compound, 151; monuron, 114; nabam, 152; parathion, 62; picloram, 119; quinine, 154; TCA, 122; thiram, 155; toxaphene, 71; sodium chlorate, 120; zectran, 75; ziram, 156
Chemical constituents in bean and corn plants, 13, 27, 43-44, 65-66, 178-180. *See also* Bean plant; Corn plant
Cherry tree. *See* Black cherry tree; Chokecherry tree
Chickadee, residues in, 106
Chicken: chick mortality, 14, 17, 51, 55, 68; egg abnormal 125, 170; egg yield reduced, 1, 58, 113, 125, 126, 127; fertility depressed, 125, 170; fertility unchanged, 58, 113, 126, 127; hatchability, 58, 113, 126, 126, 127; LD_{50}, 58, 107, 113, 126; neurotic syndrome in, 14; poisoned, 141; residues in 101; unaffected, 50; weight loss, 14, 58, 125, 126; weight unchanged, 58, 113, 127
—— embryo mortality caused by: aldrin, 4; binapacryl, 9; carbaryl, 10; chlorbenside, 12; chlordane, 13; demeton methyl, 30; dichlorvos, 31; dicofol, 33; dieldrin, 34; DNOC, 40; endosulfan, 41; endrin, 42; ethion, 43; heptachlor, 47; lindane, 51; malathion, 53; mecarbam, 55; menazon, 55; methoxychlor, 56; monocrotophos, 58; nicotine, 60; parathion, 61; phosphamidon, 65; rotenone, 67; schradan, 68; TDE, 68; toxaphene, 71; trichlorfon, 74
—— teratogenesis in, caused by: captafol, 126; captan, 126; carbaryl, 10; demeton methyl, 30; ethion, 43; folpet, 147; malathion, 53; mecarbam, 55; menazon, 55; monocrotophos, 58; nicotine, 60; parathion, 61; phosphamidon, 65; schradan, 68
—— weakness in legs of, caused by: abate, 3; azinphos-methyl, 8; carbaryl, 10; carbophenothion, 12; Chlordrin, 13; coumaphos, 15; crufomate, 16; DEF, 100; dicapthon, 31; dioxathion, 38; disulfoton, 39; dursban, 40; EPN, 43; ethion, 43; fenthion, 46; malathion, 53; menazon, 55; merphos, 114; mobam, 56; parathion, 61; phorate, 64; ronnel, 67; thiram, 155
Chinook salmon: biological concentration in, 101; EC_{50}, 100; LC_{50}, 21; residues in, 105; survival, 135
Chinstrap penguin, residues in, 105
Chipmunk: populations reduced, 53; populations unaffected, 16
Chironomid: poisoned, 104; populations decreased, 27, 98, 106, 115, 122; populations increased, 5, 105, 121; populations unaffected, 120
Chokecherry tree, aerial portions unaffected, 98
Chorus frog, LC_{50} to: carbophenothion, 12; 2,4-D, 98; DDT, 24; dieldrin, 35; endrin, 42; lindane, 51; malathion, 54; methoxychlor, 56; naled, 59; paraquat, 116; parathion, 62; piperonyl butoxide, 63; silvex, 132; TDE, 68; toxaphene, 72
Chub, biological concentration in, 97
Chukar partridge, LD_{50} to: abate, 3; azinphos-methyl, 8; demeton, 30; dicrotophos, 32; dieldrin, 35; dursban, 40; EPN, 43; fenthion, 46; gardona, 47; monocrotophos, 58; oxydemeton-methyl, 60; parathion, 61; phorate, 64; phosphamidon, 65; propoxur, 66; TEPP, 69; zectran, 75

Cladoceran. *See* Waterflea
Clam: egg development inhibited, 5, 10, 72, 102; mortality, 91, 115; populations, 97, 98, 116; residue in, 100; unaffected, 98. *See also* Atlantic clam; Cockle clam
Clover mite population increased, 24
Coccinellid: activity depressed, 97, 170; growth depressed, 97, 170; mortality, 90, 140, 150; oviposition, 90, 72; populations reduced, 24, 90, 95, 97; repellence of, 95; resistance in, 95; unaffected, 100, 141
Cockle clam, EC_{50}, 10
Cod, residue in, 106
Codistillation of DDT, 98
Coeur d'Alene chipmunk populations unaffected, 10
Coho salmon: fry mortality, 22, 95; LD_{50}, 21; residues in, 22, 99, 109
—— LC_{50} to: azinphos-methyl, 5; carbaryl, 10; DDT, 20; dicamba, 102; diuron, 105; fenthion, 40; lindane, 52; malathion, 58; parathion, 62; picloram, 119; rotenone, 67; toxaphene, 71; zectran, 75
Coleoptera. *See* Beetle
Collembola. *See* Springtail
Columbian ground squirrel populations unaffected, 10
Concord grape damaged, 97, 99
Coot reproduction reduced, 13, 71
Copepod: mortality, 140; populations increased, 141
Copper content of plants, 44
Corixid, no mortality, 41
Cormorant, residues affecting eggshells of, 15. *See also* Brandt's cormorant; Double-crested cormorant
Corn plant: attractive to mouse, 90; copper content of, 44; growth, 37, 40, 170; iron content of, 44; residues in, 5, 8, 171
Corvid, residues in, 164
Cottid mortality, 29
Cotton plant growth, 90
Cotton rat reproduction, 9
Cottontail rabbit, repellence of, 90, 98, 140–141, 155
Cottonwood mortality, 115
Cottony-cushion scale population outbreak, 58, 85
Coturnix: ataxia in chicks, 16; carbonic anhydrase activity, 16; egg production, 7, 16; eggshells, 7, 16–18; fertility, 16; hatchability, 16; mortality, 7, 17; residues in, 84; weight, 16–18
—— LC_{50} to: abate, 3; aldrin, 4; amitrole, 86; Aroclor, 7; azinphos-methyl, 5; cadmium succinate, 156; captan, 135; carbaryl, 9–10; chlordane, 12; coumaphos, 15; 2,4-D, 98; dalapon, 100; DDE, 16; DDT, 16; demeton, 29; dichlobenil, 103; dicofol, 32; dieldrin, 36; dimethoate, 35; diquat, 104; disulfoton, 37; diuron, 105–106; dursban, 40; endosulfan, 41; endrin, 42; EPN, 44–45; fenac, 109; fenthion, 46; fenuron, 110; heptachlor, 47; lindane, 51; malathion, 57; mercury compound, 150; methoxychlor, 60; mirex, 58; monuron, 114; nabam, 152; naled, 59; parathion, 61; Paris green, 64; perthane, 51; phosphamidon, 65; silvex, 121; simazine, 123; 2,4,5-T, 126; TDE, 66; terpene polychlorinates, 70; tetradifon, 70; toxaphene, 71; trichlorfon, 74
—— LD_{50} to: abate, 3; carbaryl, 9; 2,4-D, 98; DDT, 16; demeton, 29; dicrotophos, 32; dieldrin, 36; dioxathion, 39; dursban, 40; EPN, 44; fenthion, 46; mercury compound, 150; monocrotophos, 59; nabam, 152; nicotine sulfate, 60; oxydemeton-methyl, 60; parathion, 61; PCP, 117; propoxur, 66; zectran, 75
Cow: attracted to ragwort, 95; nitrate level toxic in, 100; ragwort toxic in, 100; repellence of, 95, 107, 117
Cowbird: residue in, 17, 90; DDT kinetics in, 17
Crab: LC_{50}, 11; paralyzed, 11; populations, 98, 99. *See also* Blue crab; Dungeness crab; Fiddler crab; Hermit crab; King crab
Crab-eater seal, residues in, 169
Cranberry, no residues in, 170
Cranberry bog, persistence in, 104
Crane. *See* Lesser sandhill crane
Crawfish. *See* Red crawfish
Crayfish: biological concentration in, 26; in diet of fish, 20; unaffected, 55, 126
Creeping thistle, resistance in, 100
Crested auklet, residues in, 94
Cricket, residue in, 6
Cricket frog, resistance in, 25
Croaker. *See* Atlantic croaker
Cross-resistance in mosquito fish, 5, 75
Crustacean, biological concentration in, 151; populations eliminated, 98
Cucumber, translocation into, 5, 98, 99
Cutthroat trout: mortality, 21, 22; unaffected, 99

Dace unaffected, 22
Damselfly populations, 95, 115, 116, 125, 126, 128
Daphnia. *See* Waterflea
Decomposition of: cellulose, 99; litter, 11
Deer: browse improved, 98; residues in, 140, 164. *See also* Mule deer
Degradation of pesticides, 5, 98, 100, 101, 116, 140
Development inhibited, 95, 96, 97. *See also* Egg development; Growth; Weight
Diabetes syndrome, 75
Diatom production, 98
Diet change, 22, 26, 95
Diffusion from seed coat to fruit, 101
Diptera: biomass reduced, 5, 20; populations decreased, 26, 95, 129; populations increased, 95, 98, 102, 123–124; populations unaffected, 5. *See also* Biting midge; Chironomid; Horsefly; Housefly; Mosquito; Phantom midge
Disease: diabetes syndrome, 75; duck hepatitis virus, 7, 130; susceptibility to, 130
Diversity reduced, 26
Dog, LD_{50} to: chloroxuron, 91; 2,4-D, 98; DDT, 16; dieldrin, 36; DMPA, 107; fluometuron, 111; 2,4,5-T, 126; toxaphene, 70; trifluralin, 129; zectran, 75
Double-crested cormorant: eggshell affected by residues, 15; residues in, 106, 167
Dove, LD_{50}, 46, 55, 66, 75. *See also* Ringdove
Downy rabbitbrush, aerial portions affected, 98
Dragonfly: populations decreased, 108, 115; populations increased, 116, 125; populations unaffected, 96, 125
Duck, residues in, 164, 165, 167, 168. *See also* Mallard duck
Dungeness crab: hatchability, 11; LC_{50}, 11; residues in, 165

Eagle, residues in, 130. *See also* Bald eagle; Golden eagle
Earthworm: biological concentration in, 26, 36–37, 160; LD_{50}, 9; residues in, 8, 16, 169–170; survival, 46

—— mortality from: captan, 189; carbaryl, 10; 2,4-D, 97; dibromochloropropane, 31; malathion, 58; simazine, 155; DNOC, 155
—— populations: increased, 53, 123–124; unaffected, 7, 20, 53, 101, 117; decreased by: aldrin, 3; atrazine, 90; chlordane, 13; chlorpropham, 91; DDT, 20; dieldrin, 33; DNOC, 40; heptachlor, 49; metacide, 53; monuron, 115; parathion, 60; propham, 120; rotenone, 67; TCA, 120
Eastern oyster: biological concentration in, 18, 27, 37, 44, 90; mortality, 93; residues in, 94; unaffected, 93, 98
—— shell growth depressed by: DDT, 20; diuron, 108; EPTC, 109; ferbam, 147; monuron, 115; neburon, 119; prometryne, 120; silvex, 130
—— shell growth unaffected by: ametryne, 95; atrazine, 97; 2,4-D, 98; dalapon, 100; diquat, 104; MCPA, 118; paraquat, 118; picloram, 118; prometone, 119; 2,4,5-T, 127
Eel: LC_{50}, 188; population eliminated, 188
Egg: development inhibited, 5, 10, 78, 169; fertility, 53; laying delayed, 18; mortality, 80; production, 7, 18, 33, 48. *See also* Chicken, egg yield; residues in, 33, 38, 160, 161, 165–166, 178. *See also* Eggshell; Hatchability
Eggshell: abnormally shaped, 166, 170; calcium in, 18; residues affecting thickness of, 17–18, 167; soft, 166, 170; thinning, 7, 17–20, 34–36, 63, 177, 178; weight, 17–19
Eider duck, residues in, 164
Elk, residues in, 164
Elm susceptibility, 118
Embryo mortality in mink, 10, 18, 170. *See also* Chicken embryo mortality
Emperor penguin, residues in, 165
Enchytraeid, populations, 5, 26, 123–124
English sole: LC_{50}, 10; residues in, 165
Ephemeroptera. *See* Mayfly
Erythrocyte count, 18
Estradiol in blood, 18
Estrus, 5
European corn borer, population outbreak, 30
European oyster, residues in, 34
European red mite populations, 24–25
Eye cataracts, 47

Falcon, residues in, 150. *See also* Peregrine falcon; Prairie falcon
Fathead minnow: biological concentration in, 28, 44; mortality, 143; unaffected, 91, 113
—— LC_{50} to: azinphos-methyl, 8; carbaryl, 10; DDT, 20, 21; diquat, 105; endothall, 106; endrin, 42; fenthion, 45; lindane, 52; malathion, 55, 56; parathion, 62; picloram, 118; propoxur, 66; silvex, 121; TEPP, 69; toxaphene, 71; trichlorfon, 74; zectran, 75
Feathers, residues in, 165
Feeding rate, 26
Fertility. *See* Reproduction
Fiddler crab: behavioral change, 20; LC_{50}, 65
Field mouse, repellence of, 156
Finch: mortality, 150; residues in, 150, 164. *See also* Bengalese finch; House finch
Fin erosion, 147
Fire ant unaffected, 48
Fir foliage, residues in, 171
Fish: biological concentration in, 34, 65, 69; diet, 25, 27; populations reduced, 59, 69; residues in, 29; susceptibility to microsporidian parasite, 10, 69, 160
—— persistence in, of: azinphos-methyl, 8; 2, 4–11, 98; diazinon, 30; dieldrin, 33; diquat, 104; dursban, 41; endothall, 106; heptachlor, 49; lindane, 51; malathion, 54; parathion, 62; simazine, 125; sodium arsenite, 124
Flounder, residues in, 166
Fly. *See* Diptera
Food chain: biological concentration in, 19, 131, 150; effect on, 119
Forbs, production of, 98; residues in, 171
Forest environment, effects on, 10, 26, 45–46, 65, 66
Fork-tailed petrel, residues in, 167–168
Fowler's toad, LC_{50} to: aldrin, 3; azinphos-methyl, 8; DDT, 20; DEF, 102; dieldrin, 33; endrin, 42; heptachlor, 49; lindane, 51; malathion, 54; methoxychlor, 59; molinate, 114; paraquat, 116; silvex, 122; TDE, 68; toxaphene, 72; trifluralin, 129
Fox, residues in, 149–150
Foxtail, residues in, 6
Freshwater mussel: biological concentration in, 30; unaffected, 92
Frog: development, 95; LC_{50}, 84, 154, 156; dose lethal to, 127, 128, 150, 154, 157; mortality, 130, 142; populations, 29, 92, 112; resistance in, 150. *See also* Bullfrog; Chorus frog; Cricket frog
Fruit-tree red spider, egg production, 25
Fulmar, residues in, 164
Fulvous tree duck, LC_{50}, 4, 11–13, 20, 31, 71, 150
Fungi imperfect growth, 128
Fungus: degradation of picloram, 117; growth reduced, 148; increased, 157; mortality, 155; unaffected, 119, 134
Fur seal, residues in, 165

Gannet. *See* Atlantic gannet
Garden Turf, microarthropods in, 118
Garter snake, residues in, 6
Gestation period, mortality during, 16
Ghost shrimp, LC_{50}, 11
Gills: concentration in, 131; damaged, 106; residues in, 165
Goat: residues in, 164
—— LC_{50} to: demeton, 29; dieldrin, 33; disulfoton, 39; dursban, 40; endrin, 42; monocrotophos, 59; nabam, 166; parathion, 61; propoxur, 66; toxaphene, 70; zectran, 75
Golden eagle: LC_{50}, 30; reproduction, 34; residues in, 34, 165, 167
Golden shiner: LC_{50}, 4, 33, 40–41, 72; mortality, 114; resistance in, 4, 33, 40–41, 43, 72
Goldfish: biological concentration in, 78; growth, 119; mortality, 107, 141, 145–148
—— LC_{50} to: aldrin, 4; azinphos-methyl, 8; BHC, 51; carbaryl, 10; chlordane, 13; DDT, 20; dieldrin, 33; endothall, 106; fenthion, 46; heptachlor, 49; lindane, 52; malathion, 55; methoxychlor, 58; parathion, 62; picloram, 118; toxaphene, 71; zectran, 75
—— no mortality from: biuron, 137; carbolic acid, 150; hippuric acid, 148; maleamic acid, 140; methionine, 152; naphthalenesulfonic acid, 155; phenetidine, 158; safrole, 154
Goose. *See* Canada goose

Gopher. *See* Pocket gopher
Grape injured, 97, 98
Grass: production, 99, 98; residues in, 171
Grasshopper: growth, 97; populations, 97
Grass shrimp LC_{50} in: aldrin, 5; DDT, 24; dichlorvos, 32; dieldrin, 36; dioxathion, 39; endrin, 43; heptachlor, 49; lindane, 52; malathion, 54; methoxychlor, 57; mevinphos, 58; parathion, 60
Gray partridge: arthropods in habitat of, 110–111; LD_{50}, 36, 61
Great blue heron, residues in, 166
Great crested grebe, residues in, 166
Grebe, residues in, 155, 167–168
Green sunfish: LC_{50}, 4, 36, 40–41, 48, 72, 118; resistance in, 4, 36, 40–41, 48, 72
—— survival in concentration of amitrole, 90; atrazine, 87; 2,4-D, 94; dalapon, 100; dichlobenil, 102; endothall, 108; fenuron-TCA, 111; monuron, 114–115; paraquat, 116; silvex, 122; 2,4,5-T, 127
Green white-eye mortality, 62
Grey seal, residues in, 168
Ground beetle: biological concentration in, 36–37; feeding rate, 26; populations reduced, 26, 29; residues in, 6
Grouse, unaffected, 10. *See also* Blue grouse; Prairie sharp-tailed grouse; Ruffed grouse; Sharp-tailed grouse
Growth: stimulated, 21, 27, 40, 97, 98, 108, 170. *See also* Development; Weight
—— depressed in: aerobic organisms, 90; algae, 11, 92; bluegill, 46, 56, 100; coccinellid, 170; eastern oyster. *See* Eastern oyster, shell growth; fungus, 135; oyster, 72; plants, 26–27, 48, 49, 97, 170; rainbow trout, 55; turkey, 8; white-tailed deer, 88, 170
—— unchanged in: algae, 110; bacteria, 89, 112; eastern oyster. *See* Eastern oyster, shell growth; fungus, 110, 124; goldfish, 110; grasshopper, 97; phytoplankton, 97; waterflea, 110, 119
Guchi fish: behavioral change in, 117; LC_{50}, 125
Guillemot, residues in, 64
Guinea pig, LD_{50} in: Aroclor, 7; 2,4-D, 95; dalapon, 100; DDT, 16; dichlorophen, 144; DMPA, 107; endrin, 42; lindane, 51; malathion, 53; parathion, 61; picloram, 118; pyrethrins, 66; silvex, 121; toxaphene, 70
Gull: biological concentration in, 27–28; residues in, 166, 167
Guppy: behavioral change in, 48; LC_{50}, 86, population eliminated, 130; reproduction, 48; tolerance in, 21; unaffected, 119

Habitat altered, 178
Haddock, residues in, 166
Hake, residues in, 166
Halibut, residues in, 166
Hardness of water affecting LC_{50}, 141
Hare, residues in, 140, 168
Harlequin fish, LC_{50} in: 4-amino-3,5-dichloro-benzaldoxime, 86; AMS, 87; asulam, 87; atrazine, 87; azinphos-methyl, 8; barban, 89; benazolin, 89; bromophos, 9; bromoxynil, 89; busan, 137; captafol, 138; carbaryl, 10; carbolic acid, 139; carbophenothion, 12; chlorfenvinphos, 14; chloropropylate, 14; chlorthiamid, 92; coumaphos, 15; 4-CPA, 93; 2,4-D, 94; demeton methyl, 20; diallate, 102; diazinon, 20; dichlobenil, 102; dichlorophen, 144; dichlorophenthion, 21; dichlorvos, 21; dieldrin, 33; dimetan, 37; dinoseb, 145; dinitol, 38; diquat, 103; DNBP, 107; DNOC, 140; endosulfan, 41; endothall, 109; ethion, 45; fentin acetate, 140; formothion, 47; paraformaldehyde, 146; heptachlor, 49; ioxynil, 111; lenacil, 111; lindane, 52; malachite green, 140; malathion, 53; menazon, 56; mevinphos, 57; mirex, 58; nabam, 146; N-methyl carbamate, 60; paraquat, 116; phenoxytol, 154; phorate, 64; phosalone, 65; picloram, 118; pyrazon, 121; silvex, 121; SMDC, 155; sodium chlorate, 123; sodium nitrite, 158; 2,4,5-T, 126; trifluralin, 129; vamidothion, 74; zinc chloride, 76
Harp seal, residues in, 168
Harvest mouse, repellency of, 156
Hatchability: reduced, 17, 19, 34, 90, 126, 130; unchanged, 11, 99, 112, 126, 127
Hawk, residues in, 166
Hemiptera. *See* Bug; Corixid; Mirid; Waterbug
Hemoglobin, 10
Hepatitis virus in mallard ducks, 7, 146
Hermit crab, LC_{50} in: aldrin, 5; DDT, 24; dichlorvos, 32; dieldrin, 36; dioxathion, 39; endrin, 44; heptachlor, 49; lindane, 52; malathion, 54; methoxychlor, 57; mevinphos, 58; parathion, 60
Heron, residues in, 155, 166, 167, 168
Herring, residues in, 166
Herring gull: mortality, 30; reproduction, 17–18, 30, 34–35; residues in, 165, 166
Homoptera populations reduced, 11, 28. *See also* Aphid; Cottony-cushion scale
Honeybee: habitat improved, 30; LD_{50}, 96, 103, 124; mortality, 10, 55, 96, 112; residues in, 55; resistance in, 35–36
Hooked mussel, residues in, 24
Hornfly, populations reduced, 67, 98
House finch, LD_{50}, 30, 32, 46, 59, 66, 72
Housefly, ovarian development of, 24
House sparrow, mortality, 17
—— LD_{50} in: abate, 3; cyano(methylmercuri)guanidine, 142; demeton, 20; dicrotophos, 32; dieldrin, 35; dursban, 40; fenthion, 46; monocrotophos, 59; oxydemeton-methyl, 60; parathion, 61; propoxur, 66; zectran, 75
Hydrocyanic acid: content of plants, 99, 118, 127, 130; toxicity to sheep, 98
Hydrophilid unaffected, 41
Hymenoptera, populations reduced, 28. *See also* Fire ant; Honeybee; Leafcutting bee; Wasp

Insect: biological concentration in, 151; populations, 98, 118
Iron: content of plants, 44
Isopod unaffected, 175

Jackdaw mortality, 151
Japanese shellfish sensitivity, 117
Japanese stork, residues in, 150
Jardine's babbler mortality, 62
Jimson weed palatability, 98
Jumping mouse populations unaffected, 10
Junco, LD_{50}, 66

Kale, residues in, 170
Katla fish, unaffected, 94

Kestrel, residues in, 104, 107
Kidney: concentration in, 151; damaged, 151
Killifish: LC50, 91, 111; mortality, 88. *See also* Longnose killifish
Kinetics of DDT loss, 99
King crab, residues in, 100
Kittiwake, residues in, 104
Kurrichaine thrush mortality, 89

Ladybird beetle. *See* Coccinellid
Lake habitat: biological concentration in, 97-98, 99, 78; bottom fauna reduced, 79
Lake chub-sucker survival in concentration of: amitrole, 90; dalapon, 100; dichlobenil, 100; diquat, 104; endothall, 108; fenac, 109; fenuron-TCA, 111; monuron, 114-115; silvex, 122
Lake emerald shiner: LC50, 93, 94, 105, 108, 122, 124; unaffected, 149
Lake trout: fry mortality, 22, 178; reproduction stopped, 23; residues in, 160, 178
Lambsquarter, nitrate content of, 96
Landlocked salmon, LC50, 80
Largemouth bass: blindness in, 146-147; mortality, 94, 141, 142, 144; residues in, 99, 100; unaffected, 99, 108, 122
—— LC50 to: azinphos-methyl, 8; carbaryl, 10; DDT, 20; diquat, 105; diuron, 106; endothall, 109; fenthion, 40; lindane, 50; malathion, 58; parathion, 62; toxaphene, 71; zectran, 75
Leafcutting bee mortality, 9, 10, 20
Leech: populations reduced, 88, 99, 123; unaffected, 129
Lepidoptera populations reduced, 28. *See also* Caterpillar; European corn borer; Rice stemborer
Legume nodulation, 118
Lesser sandhill crane, LD50, 16, 40, 60, 71, 76
Lesser scaup, residues in, 105
Lettuce yield reduced, 93
Leukocyte count, 16
Litter decomposition, 11
Little egret, residues in, 100
Little neck clam, residues in, 160
Little owl, residues in, 105
Liver: concentration in, 151; damaged, 7, 99, 122, 124; residues in, 16, 150-151, 155, 157-158
Livestock, repellence of, 93
Long-eared owl mortality, 84
Longevity of aphids,
Longnose killifish: LC50, 16, 115, 146, 149; mortality, 89; unaffected, 80, 100, 104, 116

Mackerel, residues in, 105
Malaria control, 25
Malformation of plant, 97, 99
Mallard duck: disease in, 7, 75, 150; egg production, 7; eggshell thinning, 7, 16, 24, 92; embryo mortality, 16, 170; reproduction depressed, 88, 90, 95, 165, 131, 170; residues in, 104, 107, 160; unaffected, 7, 40, 46, 69, 124
—— LC50 to: abate, 3; aldrin, 4; amitrole, 90; Aroclor, 7; atrazine, 97; azinphos-methyl, 8; captan, 130; carbaryl, 9-10; chlordane, 12; 2,4-D, 98; dalapon, 100; DDE, 18; DDT, 18; demeton, 26; dichlorvos, 31; dicofol, 32; dieldrin, 34; dimethoate, 36; diquat, 104; disulfoton, 35; diuron, 105-106; endosulfan, 41; endrin, 42; fenac, 109; fenitrothion, 40; fenthion, 40; fenuron, 110; heptachlor, 47; lindane, 51; malathion, 55; mercury compound, 130; methoxychlor, 58; monuron, 114; nabam, 132; naled, 59; parathion, 61; Paris green, 64; perthane, 64; phorate, 65; phosphamidon, 66; picloram, 116; simazine, 120; 2,4,5-T, 120; TDE, 69; terpene polychlorinates, 70; tetradifon, 70; toxaphene, 71; zectran, 75
—— LD50 to: abate, 3; aldrin, 4; allethrin, 5; aminocarb, 6; amitrole, 90; atrazine, 97; azinphos-methyl, 8; captan, 99; carbaryl, 9; carbofuran, 11-12; chlordane, 12; chloroxuron, 97; chlorpropham, 91; copper sulfate, 131; coumaphos, 13; cycloheximide, 132; cyanomethylmercuri guanidine, 132; 2,4-D, 98; DDT, 19; demeton, 26; diazinon, 30; dibromochloropropane, 30; dichlobenil, 100; dichlone, 132; dichlorvos, 31; dicloran, 132; dicrotophos, 32; dieldrin, 33; dimethoate, 36; dioxathion, 38; diquat, 104; disulfoton, 37; diuron, 105; DNBP, 38; dursban, 40; Dyrene, 140; endosulfan, 41; endrin, 42; EPN, 41; fenitrothion, 40; fenthion, 40; fenuron, 111; folpet, 147; gardona, 47; heptachlor, 47; lindane, 51; malathion, 55; mercury compound, 130; methomyl, 56; methoxychlor, 58; mevinphos, 57; mirex, 58; monocrotophos, 59; nabam, 132; naled, 59; nicotine sulfate, 60; oxydemeton-methyl, 60; parathion, 61; phorate, 64; phosphamidon, 65; picloram, 116; propoxur, 66; pyrethrins, 66; rotenone, 67; schradan, 68; TEPP, 69; thiram, 136; toxaphene, 70-71; trifluralin, 123; zectran, 75; ziram, 137
Mallow palatability, 98
Manganese content of bean plant, 97
Manure worm. *See* Earthworm
Maple. *See* Box elder
Margarodid. *See* Cottony-cushion scale
Marsh habitat, 33, 71
Marten, residues in, 149-150
Mayfly: EC50, 28, 35, 45, 49; LC50, 54, 73; unaffected, 95, 125
—— populations reduced by: aldrin, 3; atrazine, 97; 2,4-D, 98; DDT, 25, 26; diuron, 106; monuron, 115; neburon, 116; simazine, 122
Meadowlark, repellence of, 148, 150
Melba finch mortality, 62
Mesostigmatid mite populations reduced, 26
Methylation by microorganisms, 131
Microbial degradation. *See* Degradation
Microorganisms: activity suppressed, 10, 117, 126; composition changed, 112; numbers increased, 98; unaffected, 99, 99, 99, 112, 120
Microsporidian parasite, 10, 95, 160
Midge. *See* Chironomid
Milk, residues in, 163
Millet unaffected, 11
Millipede populations, 28, 101, 115, 128
Mink: adrenal weight, 16; births reduced, 16; embryo mortality, 16; erythrocyte count, 16; hemoglobin, 16; leukocyte count, 16; residues in, 16, 153; spleen weight, 16
Minnow. *See* Bluntnose minnow
Mirid populations reduced, 145

Mite: resistance in, 160; unaffected, 88, 97, 115, 136, 157
—— populations decreased by: binapacryl, 9; copper oxychloride, 140; DDT, 26; dichlone, 144; dinocap, 145; monuron, 115; oxythioquinox, 61; PBB, 154; simazine, 129; sulfur, 155; thiram, 156; zineb, 158
—— populations increased by: aldrin, 5; BHC, 52; chlordane, 13; dalapon, 101; DDT, 24, 25, 26; lindane, 52; TCA, 128
Modes of action, 177
Mollusc: populations reduced, 95; unaffected, 55, 72, 117
Moorhen, residues in, 165
Moose, no residues in, 169
Mosquito: development stopped, 96; populations reduced, 87, 123
Mosquito fish: abortion in. *See* Abortion in mosquito fish; mortality, 62; preferred higher salinity, 21, 179; survival, 60
—— LC_{50} to: aldrin, 5; cacodylic acid, 80; DDT, 20, 21; dieldrin, 35; dursban, 40-41; endrin, 45; heptachlor, 49; toxaphene, 72
—— resistance in, 180; to aldrin, 4-5; chlordane, 13; DDT, 21; dieldrin, 35; dursban, 40-41; endrin, 45; heptachlor, 49; terpene polychlorinates, 70; toxaphene, 72
Moth, 25
Moulting of prawns to occur, prevented, 11
Mountain chickadee, residues in, 166
Mountain sucker mortality, 22
Mountain whitefish, residues in, 23
Mourning dove, LD_{50}, 46, 55, 66, 75
Mouse: attracted, 85; births reduced, 38, 42, 53; mortality, 19; populations decreased, 37, 45, 53; repellence of, 142, 156; residues in, 16, 42, 169; unaffected, 9, 16. *See also* White-footed mouse
—— LD_{50} to: ametryne, 82; atrazine, 97; chlordane, 12; 2,4-D, 98; dalapon, 100; DDT, 18; dibromochloropropane, 30; dichlorprop, 104; dichlorvos, 31; dicrotophos, 33; EPTC, 109; fluometuron, 111; lindane, 51; malathion, 55; methoxychlor, 58; mirex, 59; parathion, 61; picloram, 119; silvex, 121; sodium arsenite, 124; TCA, 128; TDE, 69; trifluralin, 135
Movement in the environment, 181
Mud, residues in, 17, 69, 166, 172
Mud shrimp, LC_{50}, 11
Mule deer, LD_{50} to: aminocarb, 8; carbaryl, 9; 2,4-D, 98; dieldrin, 35; dimethoate, 37; fenitrothion, 15; methomyl, 56; monocrotophos, 59; naled, 59; parathion, 61; propoxur, 66; toxaphene, 70; zectran, 75
Mullet: LC_{50}, 10, 52, 108, 114, 140; LC_{100}, 8, 62; mortality, 22, 100; skeletal damage, 62, 126
Mussel: biological concentration in, 68, 80; EC_{50}, 10; residues in, 24, 169
Myrtle warbler population reduced, 19

Nematodes: attracted, 97; populations, 5, 29, 88, 101, 119; reproduction increased, 97
Nervous syndrome in chickens, 14
Nervous system, 18, 35
Nesting success reduced, 46
Nitrate: content of plants decreased, 92, 107, 112, 118, 127; content of plants increased, 91, 95, 107, 112, 127, 130; poisoning, 91-92; production unchanged, 120, 134
Nitrification: accelerated, 98, 120, 124; reduced, 90, 92, 99, 115, 124, 138, 140, 144; unchanged, 91, 92, 109, 112, 115, 119, 153
Nitrogen content of plants, 90, 96-98, 140
Northern pike, blindness in, 146-147
Northern puffer: LC_{50}, 12; mortality, 12
Northern quahog, residues in, 24

Oak damaged, 99
Oat: aphids on, 97; nematodes on, 97
Ocean perch, residues in, 168
Odonata. *See* Damselfly; Dragonfly
Oil content in seeds, 5, 50
Old-field mouse unaffected, 9
Old-squaw duck, residues in, 165
Oligochaete: biological concentration in, 151; populations decreased, 55, 96, 116, 128; populations increased, 104, 116, 122; populations unaffected, 68, 72. *See also* Earthworm
Omnivorous bird, residues in, 164
Omycete growth reduced, 148
Onion, residues in, 51
Orchard: bird mortality in, 61, 62
—— beneficial arthropods in, affected by: captan, 138-139; copper sulfate, 141; dichlone, 144; ferbam, 147; glyodin, 148; lime sulfur, 149; sulfur, 155; thiram, 156; zineb, 158
Oregon junco, LD_{50}, 60
Oriental fruit moth, 25
Orthoptera populations reduced, 38. *See also* Cricket; Grasshopper
Osprey: eggshell weight decreased, 17-18; residues in, 165
Ostracod populations increased, 141
Outbreaks caused by insecticide use, 26
Ovarian development, 24
Oviposition: prevented, 72; rate reduced, 25
Ovulation time, 17, 179
Owl: mortality, 55; residues in, 160, 164, 165, 167
Oxygen level in water, 180
Oyster: biological concentration in, 41, 50, 53, 57, 180; EC_{50}, 10, 130; growth reduced, 5, 72; population reduced, 72; residues in, 41, 36, 169, 170. *See also* Crested oyster; Eastern oyster

Pacific oyster: EC_{50}, 10; residues in, 24, 169
Pacific spider mite, population increased, 24
Palatability, 98
Paralysis: in brown shrimp, 55, 58, 60, 101, 132; in crab, 11; in white shrimp, 129
Parasitization reduced, 93, 147, 152, 155
Parsnip palatability, 99
Partridge, residues in, 159. *See also* Chukar partridge; Gray partridge
Parula warbler population reduced, 19
Pathological lesion, 95
Pauropoda population reduced, 5
Pea: root-rot index, 149; yield reduced, 29, 51; yield unaffected, 147, 156
Peach aphid population outbreak, 69
Peanut: biological concentration in, 37, 50; residues in, 5

Pelican: biological concentration in, 73; correlation between residues and eggshell thinning in, 15. *See also* Brown pelican
Penguin. *See* Adelie penguin, Chinstrap penguin
Perch: mortality, 141; residues in, 160. *See also* Yellow perch
Peregrine falcon: eggshell weight decreased, 17–18; residues in, 165, 180
Periphyton, biological concentration in, 73
Persistence: in environment, 151; in forest habitat, 45, 46, 171; in plants, 11, 100; in ponds, 100, 104; in sediment, 100; of phytotoxic residues, 50, 101
—— in fish, of: azinphos-methyl, 8; 2, 4-D, 95; diazinon, 30; dieldrin, 35; diquat, 104; dursban, 41; endothall, 106; heptachlor, 49; lindane, 51; malathion, 54; parathion, 59; simazine, 125; sodium arsenite, 124
—— in soil, of: aldrin, 6; amiben, 85; amitrole, 86; ammonium thiocyanate, 86; AMS, 87; atrazine, 88; barban, 89; BHC, 88; cacodylic acid, 90; captan, 135; carbophenothion, 12; CDAA, 90; CDEC, 91; chloranil, 139; chlordane, 14; chlorpropham, 92; 4-CPA, 99; 2, 4-D, 100; dalapon, 101; DDT, 26, 171; demeton, 14; Dexon, 143; diazinon, 30; dicamba, 102; dichlobenil, 103; dichlorprop, 104; dieldrin, 37; Dilan, 37; dimethoate, 38; disulfoton, 39; diuron, 106; DMTT, 145; DNBP, 106; DNOC, 40; endrin, 44; EPN, 45; EPTC, 109; fenac 110; fenuron, 111; ferbam, 147; formalin, 148; heptachlor, 50; hydroxymercurichlorophenol, 149; isodrin, 50; lead arsenate, 51; linuron, 112; malathion, 55; maleic hydrazide, 113; MCPA, 113; MCPB, 114; mercury compound, 150; monuron, 115; nabam, 153; neburon, 116; parathion, 60; PCNB, 153; PCP, 117; phorate, 65; picloram, 119; prometone, 119; prometryne, 120; propazine, 120; propham, 120; Semesan, 121; silvex, 158; simazine, 124; SMDC, 156; sodium chlorate, 126; 2, 4, 5-T, 127; 2, 3, 6-TBA, 128; TCA, 128; tifourea, 130; thiram, 157; toxaphene, 73, 74; trifluralin, 130; zineb, 158; ziram, 158
—— in water, 171–172; of amitrole, 86; Cytrol Amitrole-T, 92; dichlobenil, 104; dichlorvos, 31; diquat, 105; paraxon, 61; parathion, 63; trichlorfon, 74
Petrel residues in, 160, 167–168
Phantom midge: populations decreased, 26, 90, 125; populations increased, 141
Pheasant: behavioral changes, 34; lethal dose, 40, 107; mortality, 16–17, 33, 43, 159; poisoned, 107; population reduced, 20; repellence of, 140, 149; reproduction, 33, 64, 71, 159, 170; resistance in, 177; unaffected, 40, 46, 69, 130, 160; weight loss, 71
—— LD_{50} to: abate, 3; aldrin, 4; amitrole, 86; Aroclor, 7; atrazine, 87; azinphos-methyl, 8; cadmium succinate, 135; captan, 136; carbaryl, 9–10; chlordane, 13; coumaphos, 18; 2,4-D, 98; dalapon, 100; DDD, 16; DDT, 16; demeton, 29; dichlobenil, 103; dicofol, 32; dieldrin, 33; dimethoate, 38; diquat, 104; disulfoton, 39; diuron, 105–106; endosulfan, 41; endrin, 42; EPN, 44–45; fenac 109; fenitrothion, 45; fensulfothion, 46; fenthion, 46; fenuron, 110; heptachlor, 47; lindane, 51; malathion, 53; mercury compound, 150; methoxychlor, 56; mirex, 58; monuron, 114; nabam, 152; naled, 59; parathion, 61; Paris green, 64; perthane, 64; phorate, 64; phosphamidon, 65; picloram, 118; silvex, 121; simazine, 123; SMDC, 155; 2,4,5-T, 126; TDE, 66; terpene polychlorinates, 70; tetradifon, 70; toxaphene, 71; zectran, 75
—— LD_{50} to: abate, 3; aldrin, 4; aminocarb, 6; azinphos-methyl, 8; carbaryl, 9; carbofuran, 11–12; copper sulfate, 141; *cyano(methylmercuri)guanidine*, 142; 2,4-D, 98; DDD, 16; DDT, 19; demeton, 29; diazinon, 30; dicamba, 102; dichlobenil, 103; dichlorvos, 31; dicrotophos, 32; dieldrin, 33; dioxathion, 38; dursban, 40; endrin, 42; EPN, 44; fenthion, 46; gardona, 47; mercury compound, 150; mevinphos, 57; monocrotophos, 59; nabam, 152; nicotine sulfate, 59; oxydemeton-methyl, 60; parathion, 61; PCP, 117; phorate, 64; picloram, 118; propoxur, 66; rotenone, 67; TDE 16; TEPP, 69; thiram, 156; toxaphene, 10, 71; trifluralin, 129; zectran, 75; zineb, 157
—— residues in, 164, 166, 167; in eggs, 160, 169; in fat, 30, 160; in liver, 160–161
Photosynthesis, 27
Phytoplankton: affected, 105; growth, 27, 97; mortality, 115, 116; residues in, 27, 171; unaffected, 112, 125; uptake of DDT rapid, 27
—— productivity: unchanged, 106, 118; reduced by aldrin, 5; chlordane, 13; DDT, 27; dieldrin, 36; diquat, 105; diuron, 106; Dyrene, 146; endrin, 43; ferbam, 147; folpet, 148; lindane, 52–53; methoxychlor, 57; mirex, 58; paraquat, 117; silvex, 122; toxaphene, 72
Phytotoxic residues, 50, 101
Pickerel, toxicity to, 141
Pigeon (*Columba livia*): residues in, 160, 164; steroid metabolism, 17, 33
—— LD_{50} to: abate, 3; carbaryl, 9; 2,4-D, 98; DDT, 19; demeton, 29; dicrotophos, 32 dieldrin, 33; dursban, 40; endrin, 42; EPN, 44; fenthion, 46; mercury compound, 150; monocrotophos, 59; nabam, 152; nicotine sulfate, 59; oxydemeton-methyl, 60; parathion, 61; phosphamidon, 65; propoxur, 66; zectran, 75
Pigweed, nitrate content of, 98
Pike: age affecting mercury content of, 151; concentration in, 151; mortality, 137. *See also* Northern pike; Pickerel
Pine. *See* White pine
Pine mouse, resistance in, 180
Pine squirrel populations unaffected, 16
Pinfish, residues in, 21–22, 36
Pink shrimp: immobilized, 24, 52; mortality, 52; unaffected, 89, 110, 130
Plaice, residues in, 164
Plankton: biological concentration in, 50, 73; residues in, 28. *See also* Phytoplankton; Zooplankton
Plants. *See* Vegetation
Plecoptera. *See* Stonefly
Pocket gopher: diet changed, 88; populations, 16, 88
Polar bear, residues in, 163, 164
Polecat, residues in, 149–150
Pollen: residues in, 55; toxic to honeybees, 118
Pond: biological concentration in, 28; bottom organisms in, 122; dispersion into, 65; fish in, 109; interval rates in, 109; oxygen level in, 109; persistence in, 100, 104; vegetation in, 106
Pondweed, residues in, 171
Poplar mortality, 116
Population

—— decrease, 177–178; of algae, 147; American redstart, 10; ant, 107; amphipod, 96; arthropod, 48; beetle, 26, 89; bird, 65; biting midge, 88, 98, 122; blue waxbill, 62; bottom fauna, 72; bug, 145, 157; caddice fly, 25, 96, 98, 99, 115; carabid beetle, 26, 89; catfish, 120 chipmunk, 58; clam, 96 coccinellid, 24, 25, 89, 55; common midge, 87, 98, 122; crab, 96; crustacean, 96; damselfly, 115, 125; Diptera, 25, 122; dragonfly, 115; earthworm. *See* Earthworm populations decreased; eel, 120; fish, 89, 62; fly, 25; green white-eye, 62; guppy, 120; Hemiptera, 11, 89; Homoptera, 11, 89; honeybee, 55; horsefly, 87, 98; Hymenoptera, 89; insect, 68; invertebrates, 100; Jardine's babbler, 62; Kurrichaine thrush, 62; leech, 88, 96, 122; Lepidoptera, 89; mayfly. *See* Mayfly populations reduced; melba finch, 62; mesostigmatid mite, 26; microorganisms, 99; millipede, 115; mirid, 145; mite. *See* Mite populations decreased; mollusc, 96; mosquito, 87; mosquito fish, 69; mouse, 57, 43, 58; myrtle warbler, 10; nematode, 98, 101, 112; Oligochaete, 88, 96, 122; Orthoptera, 89; oyster, 71; parasite, 71; parula warbler, 10; pauropoda, 5; phantom midge, 88, 98, 122; pheasant, 50; pocket gopher, 58; pumpkinseed, 94; quail, 33, 48; red-eyed vireo, 10; reindeer, 69; robin, 10, 58; snail, 96, 72, 99, 120, 125; songbird, 10, 48; speckled coly, 62; spider, 11, 53; springtail, 5, 98, 115, 123; stonefly, 25, 96; tree swallow, 10; wasp. *See* Wasp, parasitic, populations decreased; water beetle, 98, 122; water bug, 125; wireworm, 52, 88, 115; yellow-eye, 62; zooplankton, 125

—— increase of: aphid, 55, 90, 97; beetle, 123–124; cabbage aphid, 88; caddice fly, 5, 25; chironomid, 5, 107, 122; clam, 119; clover mite, 24; copepod, 141; cottony-cushion scale, 25, 88; damselfly, 56, 116; Diptera, 25, 55, 122, 123–124; dragonfly, 116, 122; earthworm, 62, 122; enchytraeid, 123–124; European corn borer, 90; European red mite, 24–25; grasshopper, 90; invertebrate, 26; millipede, 101, 126; mite. *See* mite populations increased; nematode, 97; Oligochaete, 106, 116, 122; ostracod, 141; Pacific spider mite, 25; peach aphid, 88; phantom midge, 141; prairie vole, 57; red-banded leaf roller, 24; red mite, 24; rotifer, 141; sarcoptiform mite, 26; snail, 97, 116; springtail, 26, 101, 126; two-spotted mite, 24; water beetle, 56; waterbug, 97; waterflea, 141; yellow scale, 25; zooplankton, 14;

—— unaffected: amphipod, 125; Atlantic salmon, 86; bacteria, 112, 101; beetle, 5, 123; black bear, 10; brook trout, 86, 124; buff-bellied chipmunk, 10; bug, 125; chironomid, 125; coeur d'Alene chipmunk, 10; Columbian ground squirrel, 10; crayfish, 86; damselfly, 125, 125; Diptera, 5; dragonfly, 125; earthworm, 7, 26, 58, 101, 117; enchytraeid, 5, 26; frog, 69; fungus, 112, 104; insect, 112; invertebrate, 112; jumping mouse, 10; leech, 125; mallard duck, 62; mayfly, 26, 125; microorganism, 65, 90, 99, 112; millipede, 58; mite, 58, 97, 116; mollusc, 72; mussel, 60; nematode, 5, 26; Oligochaete, 26, 72; pheasant, 62; phytoplankton, 125 125; pine squirrel, 10; pocket gopher, 10; prairie deer mouse, 57; rainbow trout, 124; redbacked mouse, 10; sagebrush white-footed mouse, 10; shrew, 58; snail, 125; springtail, 97, 116; wasp, 97, 158–159, 140; white-footed mouse, 10, 61; white-tailed deer, 10; wireworm, 97; zooplankton, 88, 125

Porcupine, repellency of, 98, 117

Porgy. *See* Pinfish

Porpoise, residues in, 168

Potato: amino acid content of, 98, 112; mites on, 18

Prairie chicken, LD_{50}, 150

Prairie deer mouse population, 57

Prairie falcon eggshell thinning, 18

Prairie sharp-tailed grouse, LD_{50}, 52

Prairie vole population, 57

Productivity. *See* Algae productivity; Phytoplankton productivity

Progesterone. *See* Steroid metabolism

Pronghorn antelope, residues in, 169

Protein content of plants, 97–98, 120

Protozoa activity, 99

Puffer. *See* Northern puffer

Pumpkinseed population, 94

Purple urchin, residues in, 170

Quahog. *See* Northern quahog

Quail: mortality, 48, 67; populations reduced, 33, 48; resistance in, 177. *See also* Bobwhite quail; California quail; Coturnix

Rabbit: mortality, 50, 107; no mortality, 166; repellency of, 140, 146, 148, 150, 155

—— LD_{50} to: aldrin, 5; chlordane, 12; 2,4-D, 98; dalapon, 100; DDT, 16; dieldrin, 38; endrin, 42; lead arsenate, 50; lindane, 51; malachite green, 140; molinate, 114; picloram, 115; silvex, 121; TCA, 126; trifluralin, 128. *See also* Cottontail rabbit

Radish, residues in, 51

Ragwort: attractiveness, 98; palatability, 130; sugar content of, 130; toxicity, 130

Rahu fish mortality, 94

Rainbow trout: acetylcholinesterase in, 74; behavioral changes in, 74; biological concentration in, 26, 73, 108; LD_{50}, 124; growth, 25; mortality, 111; residues in, 25; unaffected, 22, 108, 109, 116, 124

—— EC_{50} to: azinphos-methyl, 5; carbaryl, 10; DDT, 20; diazinon, 28–29; dieldrin, 35; endrin, 43; heptachlor, 49; lindane, 51; malathion, 56; toxaphene, 71

—— LC_{50} to: acrolein, 97; aldrin, 4; allethrin, 5; ametryne 95; atrazine, 97; azinphos-methyl, 5; BHC, 61; borax, 96; carbaryl, 10; chlordane, 12; chlordecone, 14; Chlorea, 91; chlorfluraeole, 91; chlorobenzilate, 14; chloronitropropane, 140; copper-8-quinolinolate, 140; cryolite, 15; 2,4-D, 94; DDT, 20, 21; diazinon, 28; dicamba, 103; dichlobenil, 109; dichlone, 14; dichlorophen, 144; dicofol, 32; dicrotophos, 32; dieldrin, 36; dimethoate, 38; dinethrin, 38; diquat, 105; diuron, 106; DNOC, 40; dursban, 40; endothall, 108; endrin, 43; fenac, 110; fenthion, 48; heptachlor, 41; LPN, 111; lindane, 53; malathion, 54; methoxychlor, 58; metiram, 152; mevinphos, 57; molinate, 114; monocrotophos, 60; Monuron, 114; mylone, 153; naphtha, 115; paradichlorobenzene, 153; parathion, 62; perthane, 64; phosphamidon, 65; picloram, 115, 116; PMA, 154; propazine, 120; pyrethrins, 66; rotenone, 121; silvex, 121; simazine, 125; sodium arsenite, 124; sodium chlorate, 125; sodium pentachlorophenate, 122; 2,4,5-T, 126; TDE, 69; terpene polychlorinates, 70; toxaphene, 71; tributyltin oxide, 151; trichlorfon, 72; trifluralin, 128, 129; trioxone, 129; urea, 129; vernolate, 130, 130; zectran, 73. *See also* Temperature affecting LC_{50} for rainbow trout

Rainwater, residues in, 173
Rainwater killifish mortality, 22
Rangeland, changes in species composition, 95
Rat: births, 47; virus in, 5; eye cataracts in, 47; repellence of, 142
—— LD50 to: abate, 8; acrolein, 88; aldrin, 3; allethrin, 6; ametryne, 87; amiben, 88; aminocarb, 9; amitrole, 90; AMS, 87; aramite, 7; Aroclor, 7; asulam, 87; atrazine, 87; azinphos-methyl, 8; barban, 90; benazolin, 90; benefin, 90; binapacryl, 9; borax, 90; bromophos, 9; bromoxynil, 90; cacodylic acid, 90; captafol, 138; captan, 138; carbaryl, 9; carbofuran, 11; carbophenothion, 12; CDAA, 90; CDEC, 90; chloranil, 139; chlordane, 12; chlorfenvinphos, 13; chlorobenzilate, 14; chloronitropropane, 140; chloropropylate, 14; chlorthion, 14; chloroxuron, 91; chlorpropham, 91; chlorthiamid, 91; Ciodrin, 15; corrosive sublimate, 142; coumaphos, 15; crufomate, 15; cryolite, 15; cycloheximide, 142; 2,4-D, 93; dalapon, 100; DCPA, 101; DDT, 16; DEF, 100; demeton, 20; Dexon, 143; diallate, 102; diazinon, 20; dibromochloropropane, 20; dicamba, 102; dicapthon, 21; dichlobenil, 103; dichlofenthion, 27; dichlofluanid, 143; dichlone, 143; dichlorvos, 21; dicloran, 144; dicofol, 22; dicrotophos, 22; dieldrin, 23; Dilan, 27; dimethoate, 27; dinocap, 145; dioxathion, 28; diquat, 104; disulfoton, 29; dithianon, 145; diuron, 105; DMPA, 107; DMTT, 145; DNOC, 29, 40; dodine, 145; dursban, 40; Dyrene, 146; endosulfan, 41; endothall, 106; endrin, 42; EPN, 44; EPTC, 109; ethion, 45; fenac, 109; fenitrothion, 45; fensulfothion, 46; fenthion, 46; fentin acetate, 146; fentin hydroxide, 146; fenuron, 110; ferbam, 146; fluometuron, 111; folpet, 147; formothion, 47; gardona, 47; glyodin, 148; heptachlor, 47; ioxynil, 111; isodrin, 50; lead arsenate, 50; lenacil, 111; lindane, 51; linuron, 111; malathion, 53; maleic hydrazide, 112; MCPA, 112; MCPB, 113; mecarbam, 55; menazon, 55; mercury compound, 149; merphos, 114; methomyl, 56; methoxychlor, 56; mevinphos, 57; mirex, 58; molinate, 114; monocrotophos, 59; Monoxone, 114; monuron, 114; nabam, 150; naled, 59; neburon, 115; nicotine, 60; ovex, 60; oxydemeton-methyl, 60; paradichlorobenzene, 150; paraoxon, 61; paraquat, 116; parathion, 61; Paris green, 64; PCNB, 152; PCP, 117; perthane, 64; phorate, 64; phosphamidon, 65; picloram, 118; piperonyl butoxide, 66; PMA, 154; prometone, 119; prometryne, 120; propazine, 120; propham, 120; propineb, 154; propoxur, 66; pyrazon, 121; pyrethrins, 66; ronnel, 67; rotenone, 67; schradan, 68; Sesone, 121; silvex, 121; simazine, 122; SMDC, 155; sodium arsenite, 124; sodium chlorate, 125; sodium pentachlorophenate, 155; 2,4,5-T, 125; 2,3,6-TBA, 126; TCA, 126; TDE, 68; TEPP, 69; terpene polychlorinates, 70; tetradifon, 70; thiram, 156; toxaphene, 70; triamiphos, 157; trichlorfon, 74; trifluralin, 128; vernolate, 129; zectran, 75; zineb, 157; ziram, 158
Razorbill, residues in, 164
Redbacked mouse populations unaffected, 18
Red-backed salamander, residues in, 160
Red-banded leaf roller population outbreak, 24
Red clover, resistance in, 97
Red crawfish, LC50 to: carbaryl, 11; DDT, 24; dimethoate, 28; endrin, 43; malathion, 54; mirex, 58; naled, 60; parathion, 63; phosphamidon, 66
Redear sunfish, toxicity to, 116
—— LC50 to: azinphos-methyl, 8; carbaryl, 10; DDT, 20; dichlobenil, 103; endothall, 108; fenac, 110; fenthion, 46; lindane, 52; malathion, 53; parathion, 62; toxaphene, 71; zectran, 75
Red-eyed vireo population, 10
Redfin shiner: LC50, 108; no mortality, 108; toxicity to, 116
Red mite population outbreaks, 24
Redside shiner: LC50, 108; unaffected, 22, 108
Red-wing blackbird reproduction reduced, 12, 71
Reforestation of tropical forest, 119
Reindeer: abortion in, 69; herd killed, 95; residues in, 140
Repellence of: birds, 40, 138; blackbird, 139, 143, 158; bobwhite quail, 130; coccinellid, 25; cottontail rabbit, 50, 95, 140-141, 155; cow, 99, 107, 117; livestock, 98; meadowlark, 140, 150; mouse, 142, 158; pheasant, 140, 149; porcupine, 95, 117; rabbit, 140, 148, 149, 155, 158; rat, 142; rodent, 141, 148, 158; woodchuck, 158
Reproduction: increased, 97, 179; affected by mercury poisoning, 150; unaffected, 21, 65, 112, 116, 126, 127. *See also* Birth; Egg; Eggshell; Hatchability
—— impaired in: bald eagle, 17; black tern, 71; blue-winged teal, 12; bobwhite quail, 71, 170; brown pelican, 20, 170; chicken, 125, 170; coot, 12, 71; cotton rat, 8; coturnix, 18; golden eagle, 34; guppy, 48; herring gull, 20; lake trout, 22; mallard duck, 36, 59, 100, 121, 170; pheasant, 28, 34, 71, 170; red-wing blackbird, 12, 71; shoveler, 12; wren, 71
Reproductive organs, residues in, 162
Reservoir, biological concentration in, 60
Residues, 160-173; correlated with eggshell thickness, 17, 19
Resistance in: black bullhead, 4-6, 21, 38, 48, 72; bluegill, 4, 20, 48, 72, 100; blue-green algae, 141; coccinellid, 25; creeping thistle, 100; frog, 180; golden shiner, 4, 23, 40-41, 48, 73; green sunfish, 4, 20, 40-41, 48, 73; honeybee, 25-26; insects, 25; mites, 25, 180; mosquito fish. *See* Mosquito fish, resistance in; mouse, 16, 43, 180; pheasant, 177; plants, 100; quail, 177; rye, 97
Respiration in bacteria, 127
Rhubarb, no residues in, 170
Rice stemborer growth stimulated, 97, 171
Ring-billed duck, residues in, 166
Ring-billed gull, residues in, 167-168
Ringdove: egg-laying delayed, 18; eggshell weight, 18; estradiol in blood of, 18
Robin: biological concentration in, 36, 180; populations reduced, 18, 36; residues in, 18
Rockfish, residues in, 160
Rodent, repellence of, 141, 148, 158
Roe deer, residues in, 160
Root rot decreased, 147
Rotifer populations increased, 141
Round-leafed mallow palatability, 98
Ruffed grouse habitat improved, 120
Russian pigweed, nitrate content of, 98
Rye: growth depressed, 98; mortality, 97; resistance in, 97; yield decreased, 99

Sagebrush: mortality, 99; residues in, 171
Sagebrush white-footed mouse populations unaffected, 18
Salamander, residues in, 160

Salinity, higher level preferred, 21, 170
Salmon: preferred warmer water, 21, 170; LC_{50}, 86, 109, 109, 114, 131, 144; residues in, 99; unaffected, 104. *See also* Atlantic salmon; Chinook salmon; Coho salmon; Landlocked salmon; Silver salmon
Salmonberry, no residues in, 170
Sand shrimp, LC_{50} to: aldrin, 5; DDT, 34; dichlorvos, 88; dieldrin, 39; dioxathion, 39; endrin, 44; heptachlor, 49; lindane, 52; malathion, 54; methoxychlor, 57; mevinphos, 58; parathion, 63
Saprophytic microorganisms, growth stimulated, 99
Sarcoptiform mite populations increased, 98
Scale. *See* Cottony-cushion scale; Yellow scale
Scarlet oak damaged, 90
Sea bird, residues in, 104
Seal, residues in, 103
Sealion, residues in, 103
Sea stick, residues in, 172
Sea urchin, residues in, 170
Sediment, residues in, 27, 100, 131, 172
Seed: diffusion from coat to fruit, 131; germination inhibited, 101
Sensitivity in: fatty animals, 32; fish, 32; insect predators and parasites, 28
Serviceberry mortality, 90
Sharp-tailed grouse, LD_{50} to: carbaryl, 9; demeton, 29; mevinphos, 57; naled, 59; parathion, 61; toxaphene, 71; zectran, 75
Sheep: behavioral changes in, 53, 178; LD_{50}, 50, 96; poisoned, 141; residues in, 107
Sheepshead mortality, 22
Shellfish: residues in, 169; sensitivity, 117
Shiner. *See* Redfin shiner; Redside shiner; Taillight shiner
Shiner perch, LC_{50}, 10
Shore bird, residues in, 103
Shore crab, LC_{50}, 11
Short-spined purple snail, residues in, 170
Shoveller reproduction stopped, 13
Shrew populations unaffected, 58
Shrimp: LC_{50}, 11; mortality, 111. *See also* Brown shrimp; Grass shrimp; Pink shrimp; Sand shrimp; White shrimp
Shrub, residues in, 171
Silver sagebrush mortality, 90
Silver salmon, behavioral changes in, 94, 126
Silverside mortality, 22
Skin change, 7
Skua, residues in, 105
Slug: biological concentration in, 98; residues in, 100, 170
Smallmouth bass survived a concentration of: amitrole, 89; copper sulfate, 141; 2,4-D, 94; dalapon, 100; dichlobenil, 103; diquat, 104; endothall, 106; fenuron-TCA, 111; monuron, 114-115; silvex, 120
Smartweed, nitrate content of, 89
Smelt. *See* American smelt
Snail: biological concentration in, 131; mortality, 20, 112; populations decreased, 30, 72, 98, 120, 130; populations increased, 97, 110; residues in, 170, 172; unaffected, 122
Snap bean, residues in, 31
Snowberry, aerial portions affected, 90
Snowbrush, aerial portion affected, 90
Soft-shell clam: mortality, 65; unaffected, 66
Soil: fauna, 53, 117; flora, 90; in food chain, 160; microbiological balance in, 156-157; persistence in. *See* Persistence in soil; residues in, 19, 35, 100, 170, 171
Sole: LC_{50}, 10; residues in, 168
Songbird: poisoned, 107; populations reduced, 10, 40; unaffected, 149
Sora reproduction stopped, 71
Soybean: affected, 110; biological concentration in, 50; residues in, 5
Sparrow: mortality, 17
Sparrow hawk: mortality, 24; residues in, 105, 107. *See also* American sparrow hawk
Spawning delayed, 94
Specificity to birds, 46
Speckled [illegible] mortality, 62
Speckled dace unaffected, 92
Spermatozoa in fish, abnormal, 122
Spider: egg production, 28; population reduced, 11, 66
Spinach yield reduced, 52
Spleen weight, 16
Spot: LC_{50}, 110, 131; mortality, 45; unaffected, 66, 67, 111, 119, 120
Spottail minnow, LC_{50}, 124
Springtail: populations decreased, 5, 58, 110, 126; populations increased, 98, 101, 126; populations unaffected, 97, 113
Squirrel populations unaffected, 16
Starling, residues in, 106-107
Starry flounder, residues in, 168
Steroid metabolism, 17, 88
Stickleback: LC_{50}, 10, 129; survival, 142
Stonefly: mortality, 132; populations reduced, 25, 36; residues in, 100; survival, 107; unaffected, 101, 117
——— EC_{50} to: azinphos-methyl, 8; DDT, 33; dieldrin, 38; endrin, 43; heptachlor, 49; lindane, 52; pyrethrins, 67
——— LC_{50} to: abate, 5; aldrin, 5; allethrin, 6; azide, 85; azinphos-methyl, 8; captafol, 138; carbaryl, 11; chlordane, 15; chloronitropropane, 140; 2,4-D, 93; DDT, 34; DEF, 108; Dexon, 140; diazinon, 30; dichlobenil, 102; dichlorvos, 82; dicofol, 53; dicrotophos, 36; dieldrin, 38; dimethoate, 38; disulfoton, 39; diuron, 107; DNOC, 40; dursban, 41; endosulfan, 42; endrin, 44; ethion, 45; fenac, 110; fenthion, 47; heptachlor, 49; lindane, 52; malathion, 54; methoxychlor, 57; mevinphos, 58; molinate, 114; naled, 59; naphtha, 115; ovex, 60; oxydemeton-methyl, 60; parathion, 63; phosphamidon, 65; picloram, 119; propoxur, 66; pyrethrins, 67; rotenone, 68; silvex, 120; simazine, 120; sodium arsenite, 124; TDE, 69; terpene polychlorinates, 70; toxaphene, 72; trichlorfon, 74; trifluralin, 123; zectran, 75
Stork, residues in, 100
Striped bass, LC_{50} to: copper sulfate, 141; diquat, 105; diuron, 108; formalin, 140; oxytetracycline, 152; potassium permanganate, 151; simazine, 122; tetracycline hydrochloride, 150; trichlorfon, 74
Striped mullet: LC_{50}, 59; mortality, 22
Sucker mortality, 22, 141
Sudan grass, hydrocyanic acid content of, 90, 112, 127, 130
Sugar content of plants, 90, 130
Sugar beet: amino acid content of, 98, 113; malformation, 98; nitrate content of, 98, 130

Sunfish, biological concentration in, 99, 102, 108. *See also* Bluegill ; Pumpkinseed ; Redear sunfish
Sunflower : palatability, 98 ; residues in, 6
Swallow populations reduced, 19
Sycamore susceptibility, 115

Taillight shiner : LC_{50}, 89 ; survival, 90
Tawny owl, residues in, 165, 167
Teal. *See* Blue-winged teal
Temperature : higher, preferred by salmon, 21, 179
—— affecting LC_{50} for bluegill to : aldrin, 4 ; azinphosmethyl, 8 ; chlordane, 15 ; dieldrin, 26 ; diuron, 108 ; endrin, 42, 43 ; lindane, 51, 52 ; malathion, 53 ; methoxychlor, 58 ; toxaphene, 71, 72 ; trifluralin, 120
—— affecting LC_{50} for rainbow trout to : aldrin, 4 ; azinphosmethyl, 8 ; DDT, 20, 21 ; dieldrin, 25 ; dursban, 40 ; endosulfan, 41 ; endrin, 42, 43 ; heptachlor, 49 ; malathion, 53, 54 ; methoxychlor, 58 ; naled, 59 ; toxaphene, 72 ; trifluralin, 120–122
Tendipedid. *See* Chironomid
Teratogenesis. *See* Chicken, teratogenesis in
Tern : reproduction, 71 ; residues in, 168
Testicular degenerative lesions, 122
Testosterone. *See* Steroid metabolism
Thistle. *See* Canada thistle ; Creeping thistle
Three-spine stickleback, LC_{50}, 10, 120
Threetip sagebrush mortality, 99
Thrush : mortality, 83 ; residues in, 164, 165
Tidewater silverside mortality, 22
Tilapia : LC_{50}, 5, 62 ; nonlethal dosage, 62
Toad : LC_{50}, 90 ; residues in, 6. *See also* Fowler's toad
Tokay grape injured, 99
Tolerance increased, 21, 42
Tomato plant : growth, 90 ; translocation in, 144 ; yield, 25, 81, 147, 155
Translocation : 5, 86, 90, 142, 144, 151
Tree swallow population reduced, 19
Trichoptera. *See* Caddice fly
Tropical forest defoliation, 110
Trout : behavioral change in, 176–177 ; biological concentration in, 80 ; mortality, 22, 107, 141 ; residues in, 22, 169. *See also* Brook trout ; Brown trout ; cutthroat trout ; Lake trout
True cod, residues in, 168
Turbidity in water affecting toxicity to fish, 72
Turbot, residues in, 168
Turkey : environment improved, 94, 125 ; growth, 4
Turnip crop yield, 23, 81, 82, 147, 155
Turtle unaffected, 28
Two-spotted mite population outbreak, 24

Urbanization as factor in bird mortality, 178

Vascular system, pathologic effects on, 96
Velella. *See* Coelenterid
Vegetables, residues in, 81
Vegetation : biological concentration in, 72, 151 ; changes in species diversity and density, 30 ; eliminated, 108 ; persistence in, 11, 108 ; residues in, 27 ; resistance in, 130
Velvet-leaf palatability, 98
Vireo population, 7
Virus, duck hepatitis, 7
Vole population, 37

Walleye, residues in, 169
Walrus, no residues in, 168
Warmouth, LC_{50}, 125
Warbler. *See* Myrtle warbler ; Parula warbler
Washington clam, residues in, 169
Wasp, parasitic : parasitization reduced, 155, 158
—— populations decreased by : binapacryl, 9 ; DDT, 23 ; dichlofluanid, 143 ; dieldrin, 30 ; dinocap, 145 ; ferbam, 147 ; lime sulfur, 149 ; metiram, 152 ; nabam, 152 ; rotenone, 66 ; sulfur, 155 ; thiram, 156 ; triamiphos, 157
—— populations unaffected by : captan, 138–139 ; copper oxychloride, 140 ; copper sulfate, 141 ; dithianon, 145 ; DN-111, 39 ; dodine, 146 ; oxythioquinox, 61 ; propineb, 154 ; zineb, 158
Water : concentration at surface, 23 ; hardness affecting toxicity, 72 ; oxygen level in, 106 ; residues in, 69, 171–173
—— persistence in, 171–173 : of amitrole, 88 ; Cytrol Amitrole-T, 92 ; dichlobenil, 104 ; dichlorvos, 31 ; diquat, 105 ; paraoxon, 61 ; parathion, 62 ; trichlorfon, 74
Water beetle populations, 26, 96, 123
Water boatman. *See* Corixid
Waterbug populations reduced, 87, 125
Waterflea : biological concentration in, 72, 150 ; growth unaffected, 118, 119 ; immobilized, 127 ; mortality, 118, 122, 142 ; populations increased, 141 ; reproduction unaffected, 118
—— EC_{50} to : aldrin, 3 ; allethrin, 6 ; amitrole, 88 ; aramite, 7 ; azinphos-methyl, 8-9 ; carbaryl, 10 ; chlordane, 13 ; chlorobenzilate, 14 ; cryolite, 15 ; 2,4-D, 96 ; dalapon, 101 ; DDT, 23 ; diazinon, 30 ; dichlobenil, 103 ; dichlone, 144 ; dichlorvos, 31 ; dieldrin, 34 ; diquat, 105 ; diuron, 106 ; endothall, 108 ; endrin, 43 ; fenac, 109, 110 ; fenthion, 46 ; heptachlor, 49 ; lime sulfur, 149 ; lindane, 52 ; malathion, 54 ; MCPA, 112 ; methoxychlor, 58 ; mevinphos, 57 ; monuron, 113 ; naled, 59 ; naphtha, 115 ; paraquat, 117 ; parathion, 63 ; phosphamidon, 65 ; propham, 120 ; pyrethrins, 67 ; rotenone, 67 ; silvex, 132 ; sodium arsenite, 124–125 ; TDE, 69 ; toxaphene, 72 ; trichlorfon, 74 ; trifluralin, 120 ; zectran, 75
—— LC_{50} to : aldrin, 3 ; allethrin, 6 ; aramite, 7 ; atrazine, 87 ; azinphos-methyl, 8 ; carbaryl, 11 ; carbophenothion, 12 ; chlordane, 13 ; chlorobenzilate, 14 ; chlorothion, 14 ; coumaphos, 15 ; cryolite, 15 ; 2,4-D, 96 ; dalapon, 101 ; DDT, 24 ; demeton, 28 ; diazinon, 30 ; dichlobenil, 103 ; dichlone, 144 ; dichlorvos, 32 ; dicofol, 32 ; dicrotophos, 33 ; dieldrin, 36 ; Dilan, 37 ; dimethoate, 38 ; diuron, 107 ; Dyrene, 146 ; endosulfan, 42 ; endrin, 44 ; EPN, 44 ; ethion, 45 ; fenac, 110 ; fenthion, 47 ; heptachlor, 49 ; lindane, 52 ; malathion, 54 ; methoxychlor, 57 ; mevinphos, 55 ; naled, 60 ; naphtha, 115 ; paraquat, 117 ; parathion, 63 ; perthane, 64 ; phosphamidon, 65 ; pyrethrins, 67 ; rotenone, 68 ; silvex, 132 ; sodium arsenite, 126 ; TDE, 69 ; thanite, 70 ; toxaphene, 73 ; trichlorfon, 74 ; trifluralin, 120 ; zectran, 75
Waterfowl : habitat improved, 109 ; residues in, 167
Water scavenger beetle. *See* Hydrophilid
Watershed habitat, 33
Waxbill mortality, 82
Weakness. *See* Chicken, weakness in legs of, 46
Weddell seal, residues in, 168
Weevil births increased, 54. *See also* Azuki-bean weevil

Weight: gain, 95, 110; loss, 14, 55, 71, 155, 156; unchanged, 98, 117, 127. *See also* Growth
Western grebe, residues in, 107–108
Western gull, residues in, 108
Whale, no residues in, 108
Wheat: damaged, 97; growth, 97; nitrogen content of, 98; protein content of, 97–98, 130
White catfish, residues in, 108
White crappy mortality, 190
Whitefish: biological concentration in, 27; residues in, 22
White-footed mouse: accumulation in, 15; populations unaffected, 16, 61; repellence of, 170; residues in, 6
White king pigeon, steroid metabolism of, 17, 23
White mullet: LC_{50} in, 10, 108, 148; mortality, 170
White oak damaged, 99
White owl, residues in, 184
White pelican: endo- and ecto-parasites of, eliminated, 71; mortality, 71; residues in, 105, 106, 107
White pine, translocation to upper stem and needles of, 142
White shrimp: mortality or paralysis, 109, 110; tolerance limit, 10, 72; unaffected, 105
White-tailed deer: fawn mortality, 55, 170; growth suppressed, 55, 170; populations unaffected, 16
White-tailed eagle, residues in, 183
White-tailed jackrabbit, repellence of, 140, 149
White-winged dove, LD_{50}, 65
Whiting, residues in, 108
Whitstable oyster, residues in, 170
Wild hare, residues in, 183
Wildlife management, 181–183
Wild parsnip palatability, 98
Wild pine mouse, resistance in, 42
Willow, aerial portions affected, 99
Wilt disease in bean plant, 105
Wireworm: damage to wheat, 97; populations, 53, 88, 115; unaffected, 97
Woodchuck: repellence of, 135; unaffected, 158
Woodcock: mortality, 46; reproduction, 10; residues in, 47–48
Wood pigeon: mortality, 4, 34, 46; residues in, 33–34, 48, 180, 185; unaffected, 150
Worm, aquatic. *See* Oligochaete

Yellow bullhead, LC_{50}, 108
Yellow-eye mortality, 62
Yellow perch, residues in, 150
——— LC_{50} in: azinphos-methyl, 8; carbaryl, 10; DDT, 20; fenthion, 46; lindane, 52; malathion, 55; parathion, 62; toxaphene, 71; zectran, 75
Yellow scale population increased, 25
Yellow-tail rockfish, residues in, 108
Yield, crop: decreased, 26, 31, 52, 156; increased, 58, 98, 147; unaffected, 147, 155

Zinc level in bean plant, 50, 130
Zoeae moulting prevented, 11
Zooplankton: biological concentration in, 29; populations, 135, 141; inhibited, 68, 72; unaffected, 63, 135, 144
Zygomycete: growth reduced, 148

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